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SOME OBSERVATIONS ON FACIAL NERVE PALSY*

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This society has suffered irreparable loss in the recent death of our distinguished fellow-otologists, Sir Charles Ballance and Arthur Duel. Their work in collaboration was of such high standard and had placed seventh nerve reconstruction on such a high plane that it seems almost a sacrilege at this date to venture the discussion indicated in the title, nor would this offering have been made save in the expectation that our revered friend, Dr. Duel, would have been here with his meticulous and pertinent comments.

These operators literally entered where angels had feared to tread, proved the possibility of inserting nerve transplant to replace the damaged one, demonstrated the value of decompression and the safety of work in the infected field. Their work has been safeguarded by the training of competent associates, and their generous service in publication is already attested by the reports of similar effort throughout the country.

The cases here discussed do not represent original work, as one was inspired by the method of Sir Harold Gillies[1] and J. Eastman Sheehan,[2] and the other patterned after the temporal muscle transplants for postauricular fissure reported by Samuel Iglauer[1a] and Ralph Almour.[3] The results of the latter were so unique and con-

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veyed such a correspondence with the socalled "reanimation" of muscular tissue, that they seemed to fit into the picture of what has been going on during the past forty years in plastic repair for seventh nerve degeneration, and particularly for that forgotten group of cases of many years' standing now considered hopeless. It is obvious that this "no man's land" will gradually pass out of existence, as no fresh cases of facial paralysis will long escape present day otologic efficiency, and the otologist unequipped with fallopian canal and petrous pyramid technic must soon step aside.

As there had been no thought that these cases might present conditions worthy of discussion, no preliminary photographs and records for "before and after" illustration were taken. They cannot, therefore, be presented as adequate case records and the surgical work presents nothing original. They are consequently simply placed under the scope of the title "some observations on facial nerve palsy."

History in this work has been made rapidly since 1895, and it is perhaps appropriate to review and analyze the present situation before presenting the cases in more detail.

The repair of facial palsy has for many decades stimulated scientific research. Earlier studies naturally centered on the etiology, physiologic and pathologic variation, and justifiable prognosis. The world owes much to Sir Charles Bell for his studies on the anatomy and physiology of the seventh nerve, published in 1830.[1] It was reserved, however, for his illustrious pupil, Sir Charles Ballance, to work out their practical application.[5] A review of the literature and its chronology shows that the most important steps in progress have been made within our own time—a period perhaps of the last thirty-five or forty years. Efforts at reconstruction were doubtless retarded by the fact that serious paralyses were so indelibly linked to accidental and faulty surgery, that the deeply buried and circuitous facial nerve canal only suggested further danger.

Two pertinent facts must have been impressive in this evolution: First, that aside from refrigeration palsy, many apparently severe traumatic cases would show spontaneous recovery in a few weeks or months; and second, with the established diagnosis of impossible spontaneous recovery, re-innervation in distal nerve segment or some equivalent must be re-established before too advanced atrophic change or irremediable facial distortion had taken place.

Certain striking figures seem to stand out in the story of advance. Sir Charles Bell, Sir Charles Ballance, and Sir Harold Gillies

on the other side; Harvey Cushing,[6] Arthur Duel,[7] Vilray Blair[8], and Eastman Sheehan in our own country, with most meritorious work by many others. While Sir Charles Ballance credits the earliest experiments on nerve suture to Flourens,[9] in 1827, the first nerve anastomosis, according to Cushing, was performed by Faure in 1898,[10] which unfortunately did not have "un resultat hereux," and one by Ballance in 1895.

There seem to have been four rather distinct epochs in this field: First, the early studies on the etiology and nature of seventh nerve function and its loss, work which has been admirably supplemented by the reviews and classifications of Burrow,[11] Bourbon,[12] Jones,[13] Leiner,[14] and others, with the brilliant laboratory research of Greenman,[15] Barron[16] and their associates.

Second. The period of anastomosis of distal segment of the injured nerve with spinal accessory, hypoglossal and other motor branches in the neck, the most serious criticism of which lay in its percentage of failure and the difficulty in avoiding associated facial movements.

Third. The adoption of fascial and muscle transplants to overcome facial distortion, which coincidentally brought out the suggestion of "reanimation" by nerve regeneration, and fourth, the epoch signalized by the transplant of nerve sections in the traumatized or degenerated segment of the fallopian canal. In this, earlier use of the external respiratory nerve of Bell, was successively changed to the intercostohumoral and then the anterior femoral cutaneous nerve, and it was demonstrated that a time period for Wallerian degeneration in transplanted nerve segment was an advantage, and that an infected field did not interdict fallopian duct surgery. The values of faradic and galvanic stimulation in diagnosis and therapeusis were evaluated.

The advances in the third and fourth periods were somewhat synchronous but with different objective. The first attempted reparation for deformity and loss of function in the field of distal degeneration; the latter aimed at restoration at the source. Both types of procedure seem to have demonstrated the findings of parallel research in nerve regeneration, and it is to invite comment upon their application to the so-called incurable group that this brief contribution is given.

The two patients presented for discussion present certain points in common. Both had a total unilateral seventh nerve palsy of

approximately twenty-five years' standing, following the mastoid operation. The hospital records in each case were very meager. We know time of operation, hospital and operator, all of which are inconsequential, but have very little detail of procedure. Both were recently examined by Dr. Ornsteen in the University neurologic service, and one also by Dr. Frazier. They reported an advanced form of degeneration with probable atrophy and fibrosis. No faradic and practically no galvanic response was obtained. Both presented marked facial deformity. Both cases were discussed with Dr. Duel, who considered surgical help without probable value and advised against it. Only salient points will be reviewed in these cases and they will be later shown on the screen.

REPORT OF CASES

CASE 1.—This patient, J. M., a young man of 23, was referred for examination in April, 1933, with total facial palsy on the left side. The reference was not on account of the facial weakness, but because of a large postauricular defect, oval in shape, about 2½x2 cm. in measurement, with practically no dermatization—a slight amount of offensive secretion in the depth of the opening, and this communicated through a narrow and tortuous channel with the attic cavity. He barely caught the 512 and 1024 d.v. tones on the audiometer with that ear. First, a small lipomatous collection was evacuated behind the mastoid area. After preliminary antiseptic treatment, the postauricular opening was filled after the method described by Almour[3], by swinging down a large circular flap cut from the temporal muscle, inverting in the freshened wound, with its temporal pedicle still intact. The temporal transplant was covered in turn with undercut flaps of integument. Portions of the temporal transplant extended well down, probably 2 cm. below the lobe of the ear. The convalescence was protracted on account of suppurative discharge from the ear, and if anything the palsied droop in the face seemed worse after the operation. About four months after this plastic operation the patient noted increased movement in the face, which has steadily improved during the following two and one-fourth years. There seems to have been no possible route for this reinnervation save from the temporal muscle flap, and round beneath the auricle into the facial muscles. While the fact that the movements are associated and jumpy in character will be noted in the motion picture, the net result is a very serviceable and much less deformed face.

CASE 2.—This patient, M. W., a lady of 45, came for treatment after the death of her former otologist, in June, 1932, with a total unilateral palsy of the left seventh nerve. The face was badly distorted, left eye constantly open, hearing down about 80 per cent on that side. She came for routine cleansing of the tympanic area, which constantly collected rather offensive cholesteatomatous material in the tympanic area. After observing her at intervals for about a year, the writer was so impressed by the success of the previous case that he asked Dr. Ivy to attempt relief from the deformity and paralysis by transplanted sections of the temporal muscle after the fascial and temporal muscle graft methods of Sir Harold Gillies, Blair, Sheehan and others. Dr. Ivy's report follows: "Patient was first seen on December 14, 1933, with a complete seventh nerve paralysis following mastoid trouble about twenty-five years before. May 15, 1934, at the Graduate Hospital, under

ether, a strip of temporal muscle and fascia was turned down and brought through a tunnel beneath the skin to the corner of the mouth, where it was sutured to the orbicularis oris. A second strip was carried to the orbicularis palpebrarum, beneath the skin of the lower lid, at the inner canthus of the left eye. Some suppuration followed this operation, but after the parts healed she could almost close the left eye, and there was improvement in elevation of the corner of the mouth.

April 9, 1933, at the Graduate Hospital, under avertin, two strips of fascia lata were used, one from the upper and one from the lower lip to the temporal region, which caused further improvement. I believe there is some muscular movement on the side of the face and distinct improvement in the condition generally."

This report was written on December 9, 1935, eight months after the operation. In the four and one-half months' period since, there seems to have been a rather steady gain in muscular movement in the temporofacial area, as will be noted on the film.

It remains to clarify the principles involved in this process of regeneration. Greenman, in his studies at the Wistar Institute of Anatomy and Biology on the peroneal nerve of the albino rat, found that after crushing and wiring to cause paralysis, but leaving perineureum as the tube connecting divided nerve ends, the space within the epineureum, filled with blood or serum at the site of operation, became imbedded in new tissue, forming a bridge or capsule, which connected the proximal and distal ends. Through this, connecting bridge fibers of regenerated nerve were found to pass from the proximal to the distal segments. Following the degeneration in the operated nerve, regeneration with branching of axones took place, with great increase in numbers of medullated fibers on the proximal side of the lesion, a lesser increase on the distal side, but the latter exceeded the number on the opposite or control side. This demonstrated that it was possible for central axones to show multiplication of its fibers. It was proved by Dogliotti[17] that when one-third of the nerve fibers from the central axone innervated the muscular vesiculi, and two-thirds of the fibers were diverted to innervate other muscles, the one-third fibers showed multiplication of fibers to the extent that all of the muscular vesiculi were filled.

In this connection Duel's statement that grafts excised from nerves in which Wallerian degeneration had been allowed to take place in situ, permitted the conveyance of axones from the proximal segment through the graft to the distal segment in one-half to one-third the time required by fresh grafts, with improved quality of repair, is very significant. Sir Charles Ballance believed that in the so-called anastomosis operation there was no such thing as the anastomosis of nerves. The distal end of the nerve degenerates, and after fourteen days exists as a bundle of tubes containing the broken down

fatty elements of the nerve sheath. The axis cylinders of the central segment of the nerve slide along the tunnels, pushing aside the lipoid masses until they reach the motor end organs of the muscle or muscles which had been in connection with the fibers of the original distal segment. The new fibers thus replace the old and carry the nerve impulses which cause again the contraction of muscle fiber.

These facts may aid in the explanation for what is happening in these cases. The temporal muscle, fortunately supplied by the fifth instead of the seventh nerve, is a sturdy muscle, easily exposed, and as shown by Sheehan, with muscle strands, vessels and nerves, all following a vertical direction. Furthermore, it lends itself readily to pedicle graft formation and apparently can spare much of its innervating substance without detriment to its primary function in the face.

Whether in one or both of these patients, the long dormant and presumably atrophied fibers of the seventh nerve are utilized in any degree in the regeneration of facial movement, is entirely a matter of conjecture. The apparent impossibility of this and the cortical reeducation involved, in any case, should lend special interest to the discussion.

To draw any conclusions from two isolated cases would be embarrassing. Their present status should, however, present practical suggestions and invite further consideration in the rehabilitation of such unfortunate cases.

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XL.

BASALOMA OR SO-CALLED CYLINDROMA OF THE AIR PASSAGES*

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While the study of rare types of tumors may, as a rule, be of academic interest only, at times it may be rewarded by information that is utilitarian and easily applied in clinical practice. It may be of interest to report several cases of a rare type of neoplasm found in the respiratory passages and its adnexa, a knowledge of whose characteristic pathologic and clinical features may be of distinct service in our therapy. There have appeared sporadic reports, mainly in the European literature, of a distinct type of epithelial new growth variously termed cylindroma, endothelioma and basal cell carcinoma, that is mostly frequently found in the respiratory passages and the skin about the face, that presents histologically similar lesions, and pursues comparable clinical courses. These lesions are characterized clinically by slow growth, local progression, lack of metastases, prompt regression to irradiation, and recurrence after local excision. Histologically they present a picture of anastomosing cords of slightly anaplastic epithelial cells, closely resembling basal cells, and appearing in the cut section as round, ovoid or irregular cell nests with a central lumen.

The classification and histogenesis of these tumors have been in dispute for some time and have in the main been responsible for the confusing nomenclature. The term cylindroma was originally applied by Bilroth[1] to a tumor composed of cell nests surrounded by a hyaline ring that was found in the orbit and recurred several times after removal, although it did not metastasize. He believed, however, that it was of epithelial origin. Citelli[2] reported a tumor involving the sphenoid sinus that exhibited similar clinical and microscopic features but believed that it was endothelial in origin, as did Ferreri,[3] who described three cases of cylindroma of the maxillary sinuses. Eigler,[4] in a review of rare tumors of the upper air passages, reported four

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cases, two involving the palate, one the base of the tongue and one the larynx.

Pfeiffer[5] reported two cases involving the air passages. He believed them to be of endothelial origin, but admitted that this was difficult of proof. Barbezet[6] reported a tumor that involved the face and orbit, which he believed to be of epithelial origin from the mucosa of the nose of the accessory sinuses. Davies[7] and Stillians[8] each report a tumor involving the scalp, and both conclude that the neoplasms were of epithelial origin and of basal cell type. The subject, however, was clarified by the classical and monumental work of Krompecher[9] on the basal cell carcinomas of the skin and mucous membrane. He divided the basal cell carcinomas or "basalomas" into a solid type composed of a large cell mass, an adenoid type in which there is present some attempt at reproduction of glandular structure, and a cystic type showing cell nests with a central lumen. He believed that they originated from the basal layers of the skin and mucous membrane. He reported four tumors arising in the nose and showed that their structure was similar to those found developing in the skin. In fact, the histologic picture of basal cell growths arising from the skin may so closely resemble those arising from the mucous membrane as actually to make it impossible, from the microscopic architecture alone, to determine their origin. However, the basaloma of dermal origin is most often of the solid type, while the basaloma of the respiratory passages is predominately adenoid or cystic in type. Ewing[10] describes basal cell growths as the reticulated type, corresponding to Krompecher's solid basaloma and the adenoid type, showing a tendency towards glandular reproductions. Kaufman[11] also recognized the fact that basal cell growths may exhibit a varied histologic structure and believed that the lumen of the cell nests arose from destruction of carcinoma plugs or hyaline or mucoid material. It is apparent that the modern point of view is that these neoplasms are epithelial in origin and represent the basal cell type of growth peculiar to the mucosa of the respiratory passages. Bredlau[12] has the most recent as well as the most comprehensive review of the subject in the American literature. He reported a characteristic neoplasm arising from the nose. He concluded that these neoplasms must be viewed as basal cell growths with the addition of the descriptive terms advocated by Krompecher, namely, solid, adenoid or cystic varieties.

Grossly, the neoplasm is most often a red fleshy mass more or less firm in consistency. At times the growth may appear polypoid, and in fact has been described as a fleshy polyp. It bleeds readily

upon traumatization, an important clinical finding in most cases. A striking feature of the tumor is its capacity for extensive progression and involvement of contiguous structures. In this respect it is comparable to the rodent ulcer of the skin. Growths of the antrum readily destroy the surrounding bone, involve the ethmoids, penetrate the orbit, the base of the skull, the palate, and the anterior antral wall. In contrast to the extensive invasive properties possessed by this neoplasm is its inherent lack of metastasis. Metastasis to the neighboring lymphatic node is rarely if ever seen. Parenchymatous metastases are also infrequent. Ribbert,[13] however, found a metastatic growth in the lung secondary to an antral basaloma, while Eigler[4] found metastases in the vertebræ of the spinal column in a case of cylindroma of the epiglottis. One is able, therefore, to differentiate clinically between lympho-epithelioma and transitional cell carcinoma, which produce wide extensive metastases with but relatively little local invasion, and basaloma with its marked local extension and invasion without metastases. In common with basal cell growths of the skin, the basaloma of the mucosa grows over a long period of time, measured as a rule in years. The anaplastic growths just mentioned are of fairly rapid growth and cause exitus after a relatively short period measured in months.

Basaloma also has another biologic property in common with basal cell growths originating in the derma, namely, a relative radiosensitivity. Hofer[14] and Eigler[4] report cases that showed regression when subjected to relatively small doses of irradiation. Two of our cases showed similar evidence of radiosensitivity. It is apparent that from the clinical point of view these neoplasms possess biologic properties considered characteristic for basal cell growths of the skin and constitute one of the reasons for regarding them as basal cell growths.

Another clinical characteristic is the marked tendency towards recurrence. Most of the cases observed for an appreciable period of time after resection of the growth showed recurrence. As a rule, this took a long time, varying from six months to a year or two. It is also to be noted that recurrences were found after repeated removal, and reports of three to six recurrences after as many operations are common to several cases reported. Recurrences after irradiation are also observed with similar time intervals. The delayed recurrence is probably a clinical expression of the slow, dilatory growth of basal cell tumors.

To sum up, these growths are clinically characterized by slow growing, red, fleshy masses that are frequently fungating or poly-

poid. They are capable of extensive local progression, easily penetrating surrounding bone to involve large areas. The growth is slow and there is no metastasis to the neighboring lymph nodes. In addition there is evidence of relative radiosensitivity and marked tendency to recur after repeated removal or irradiation. Bleeding from the affected part is also frequently encountered.

Histologically the neoplasms exhibit a more or less characteristic microscopic appearance. On the whole, they are mainly composed of cell nests in a connective tissue stroma. At times the stroma may be an insignificant portion of the picture and so give rise to the appearance of a solid cell mass, the so-called solid type. More often the connective tissue stroma is seen to divide the cells into cell nests, which are variable in size and shape, most often round or oval and occasionally irregular. Frequently these cell nests appear as plexiform or anastomosing cords. Many times they show a central lumen containing débris or a pink-staining homogeneous material. The solid cell nests show abortive attempts at glandular reproduction on occasion and are, therefore, known as the adenoid variety. The cell nests containing the central lumen compose the cystic type of basaloma. The cells vary from polyhedral to columnar. They have a moderate pink-staining homogeneous cytoplasm. The nuclei are round, ovoid or polyhedral and finely granular. The stroma is made of loose mature connective tissue, as a rule highly vascular. In general the tumors are composed of anastomosing cords and nests of epithelial cells in a bed of connective tissue, many of the central lumina containing cellular débris or hyaline material. Within the limits of the variation described the histologic illustrations presented in the various publications show a remarkable similarity. The picture of cell nests containing a lumen is so characteristic that it makes the type of neoplasm easy of identification. However, when the microscopic appearance is that of the solid type, confusion with transitional cell carcinoma and round cell sarcoma is not uncommon. In fact, one of our cases was first diagnosed as a transitional cell carcinoma, and the prompt response to irradiation therapy led us to believe that we had clinical substantiation for the diagnosis. However, repeated study of later sections, as well as the fact that the patient lived over a long period of time and showed no evidence of cervical or parenchymatous metastases convinced us that we were dealing with a basaloma. The sections of tissue removed at a subsequent investigation of the antrum showed typical lumen containing cell nests.

We have recently observed three patients in whom the clinical course and histologic findings were such as to classify them as cylin-

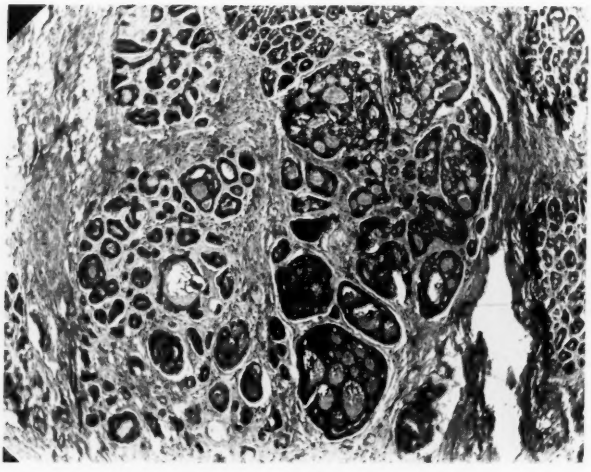

Fig. 1.

dromatous types of basal cell carcinoma of mucous membrane, or basaloma. As there is but one case, that of Bredlau, reported in the English literature, it may be of interest to describe them.

REPORT OF CASES

CASE 1. *Basaloma of the Trachea.*—Mrs. I. D., age 42, was seen on October 12, 1934, complaining of difficulty in breathing for a period of two years. Originally her dyspnea was limited to attacks that lasted for a short period of time, and was diagnosed as asthma. The dyspnea became progressively worse and for the past six months had persisted without interruption. Any degree of physical effort was impossible. Her difficulty was such that she could speak only in a whisper. Sleep was constantly interfered with by her awakening at hourly intervals with a strangling sensation.

Examination disclosed an adult half-breed Indian female, highly nervous, and able to speak only in a whisper. A marked dyspnea was present with a pronounced inspiratory crowing stridor. Speech was difficult and only a few phrases could be uttered without pausing. Her nose and throat presented no findings of interest. Her larynx was deeply injected throughout. The cords moved freely on phonation and inspiration. Her chest examination disclosed a marked inspiratory and expiratory wheeze heard throughout but most marked over the trachea. The rest of the physical examination was negative. On account of her urgent dyspnea and her state of utter exhaustion an immediate tracheotomy was decided upon.

The operation was performed under local anesthesia. It progressed without incident until the trachea lay bare ready for incision. In order to prevent the excessive cough so frequently encountered on opening the trachea, an attempt was made to inject a few drops of cocain solution into the lumen. It was surprising to note, however, that the needle did not enter an open space upon the penetration of

several millimeters. The trachea was incised but instead of the expected lumen a red firm mass was encountered. The mass was incised cautiously for about five millimeters, when the tracheal lumen was encountered. A short tracheal canula proved inadequate, but a Jackson cane canula was found to be satisfactory, as it could reach beyond the growth. A portion of the tumor was removed for histologic examination and the wound closed.

There was immediate relief from the dyspnea. In spite of the presence of the canula, to which the patient's trachea had not become tolerant, she was able to sleep through the entire night.

X-ray examination revealed a stenosis of the tracheal lumen below the level of the cricoid cartilage extending downward for about 5 cm. The stenosis was caused by apparent exterior bulge into the lumen. The lesion was posterior and unilateral. Whether this was the result of a mass intrinsic in the lumen or one projecting from behind and pushing the tracheal lumen before it, could not definitely be determined. The laryngeal ventricle could not be seen, but this may have been due to lack of air filling rather than actual pathology. A tracheotomy tube is noted extending to just above the sternoclavicular junction.

Several days later bronchoscopic examination revealed an elevated smooth red infiltrate on the right side of the tracheal mucosa. The infiltrate extended from the subglottic region above to within a few centimeters of the origin of the right bronchus. The mass occupied the right side of the trachea wall, passing beyond the midline anteriorly to invade the left side for about three to five millimeters. Posteriorly it did not quite reach the midline of the tracheo-esophageal party wall. The mucosa overlying the mass was red and congested but showed no erosion. The remaining tracheal mucosa was red and injected. The tracheal lumen showed a crescentic irregular construction of the right side. Palpation of the growth with the tip of the bronchoscope gave one the sensation of firm, almost cartilaginous tissue.

The patient recovered rapidly from her tracheotomy, but insisted upon returning to her home town for further care. She was advised to have irradiation therapy, as the extent of the growth, almost to the origin of the right main bronchus, would preclude surgical removal of the trachea. When last heard from in July, 1936, she was still alive, although leading a canula life.

The histologic examination of the tissue removed at the time of the tracheotomy revealed a tumor composed of nests of epithelial cells. The cell nests varied in size and shape with a tendency to be roughly ovoid, although many were definitely irregular in outline. The cell nests formed islands or acini and were surrounded by a rather vascular connective tissue stroma. The islands and acini were formed of compactly arranged columnar epithelial cells containing oval nuclei. The cells varied little in morphology and resembled the basal cells of the epidermis. Mitotic figures were infrequent. Many of the nests contained a central lumen in which an amorphous material was found. Diagnosis was epithelioma adenoides cysticum or cylindroma—a variety of basal cell carcinoma.

CASE 2. *Basaloma of the Antrum.*—Mrs. H. S., aged 56, was seen August 9, 1933, complaining of obstruction to breathing in her left nostril, associated with a purulent discharge. She first became aware of the difficulty in nasal respiration some two months prior to her visit, at which time she had an extensive nasal hemor-

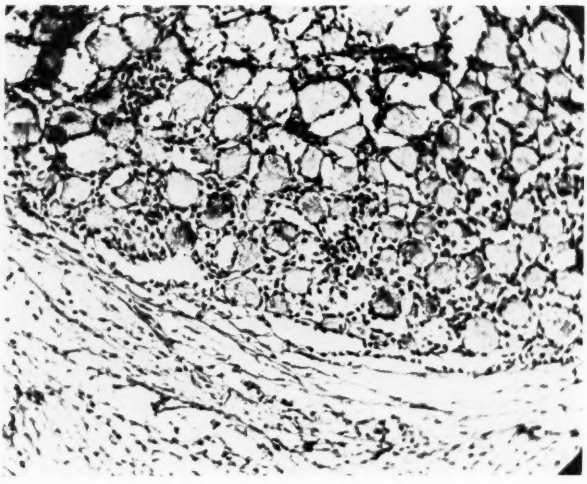

Fig. 2. Imperfect visualization due to poor staining as a result of prolonged irradiation.

rhage which required tamponage. After the pack was removed her physician found that she was unable to breathe through the left side of the nose. Her history was unimportant. Examination of the right side of the nose was negativee. The left cavity was filled with a red fungating fleshy mass that bled easily on touching with a probe. She was edentulous, the pharynx was negative and the eardrums were normal. The left side of the face was slightly swollen over the region of the canine fossa. There were no palpable glands in the neck and the rest of her physical examination was essentially negative. The left antrum was dark to transillumination, and x-ray disclosed a mass involving the entire cavity. The Wassermann reaction was negative. A sublabial exploration of the antrum was decided upon and performed on August 10th. Upon exposure of the canine fossa the tumor mass was found to lie just beneath the oral mucosa, having eroded through the anterior wall of the antrum. The tumor was soft, friable, red and fleshy in appearance. There was marked bleeding upon removing a portion for biopsy. The greater portion of the growth was resected and the cavity loosely packed. The frozen section was reported as "transitional cell carcinoma." A capsule of 25 milligrams of radium was inserted into the cavity and permitted to remain in situ fifty hours for a total dosage of 1250 milligram hours. After the irradiation the patient returned to her home town and apparently progressed very satisfactorily. Her physician kept the antral opening patent for observation and she returned for examination on September 11th, at which time there was no evidence of the growth found in the nose or in the antrum. It was decided to continue to keep the sublabial incision open for observation. She returned on December 28th, four months after the irradiation, and no evidence of new growth was present in the nose or sinus. In fact, the left side of the nose appeared normal. It was decided to let the sublabial incision close. She was not seen again until September 15, 1934, or just about a year and one month after the irradiation. At this time the nose and antral cavity were

normal to transillumination and x-ray. There were no palpable lymph nodes. On May 1, 1935, a year and eight months after irradiation, she returned complaining of bleeding from the nose. Examination disclosed no growth in the nasal cavity, but a slight streak of blood in the middle meatus of the affected side. Transillumination and x-ray disclosed that the left antral region was partly blocked. She was advised to have further irradiation and was then subjected to deep x-ray therapy. There were no glands palpable at this time. She returned home and the bleeding did not recur. She returned on December 20, 1935, because of a recurrence of her nasal bleeding. At this time there was some diminution in the vision of her left eye, which was associated with some edema of the lids. Her vision, which was formerly corrected to 20/20 in both eyes, could be corrected to but 20/40 in the left eye. There was a bleeding mass on the floor of the left nasal fossa. The left cheek was bulging somewhat. It was deemed advisable to re-expose the left antrum and insert more radium, but this was refused for the time being, as she wanted to be at home for the holidays. However, she received further deep x-ray therapy. On February 26, 1936, she returned with a definite mass presenting in the middle meatus on the left side. In addition, involvement of the palate was apparent by a bulging of the roof of the mouth. There was definite swelling over the canine fossa. On the following day the sublabial incision was reopened, and the entire cavity found to be filled with a tumor mass. As much of the growth as possible was removed and sent to the laboratory for sectioning. Histologic examination (Fig. 2) showed that the tumor was composed of epithelial cells arranged in groups, many of which had a lumen containing pink-staining amorphous material. In some portions of the growth the cells formed a compact cell mass. There was present a connective tissue stroma. The cells were definitely epithelial in character, and the diagnosis was "Cylindromatous type of basaloma."

If one viewed only a portion of the section in which the epithelial cells were in a compact cell mass, a mistaken diagnosis of transitional carcinoma could easily be made. The long drawn out clinical course, the delayed recurrences, the lack of metastases, and the slow extensive involvement, all speak for basaloma.

The patient recovered from this intervention and returned home after being subjected to further irradiation.

CASE 3. *Basaloma of the Sphenoid.*—H. L., age 51, was seen by one of us in the Otolaryngologic Clinic of the Research and Educational Hospital of the University of Illinois Medical School on September 1, 1931, and is reported through the courtesy of the Departments of Otolaryngology and Pathology.

At that time the patient complained of headache and numbness of the left side of the head, associated with impaired vision of the left eye. There was also dizziness associated with a sensation of falling that occurred on sudden movement. These symptoms had been present for about a year and a half. Several months previous the patient had been examined at another clinic, where he was informed that a mass in his left sphenoid was present. He was advised to have his tonsils removed and to have deep x-ray treatment to the tumor mass. After removal of his tonsils and the administration of two x-ray treatments his headaches disappeared, only to return some three months later. Examination of his nose showed a deflected septum with some crusting. He was edentulous, and the tonsils had been removed. Nasopharyngoscopy disclosed a tumor mass in the region of the left sphenoid and ethmoid which was firm on digital examination. The right ear was negative and the left ear showed a distorted light reflex. Transillumination of the sinus was negative, and functional examination of the right ear was normal. In the left ear the

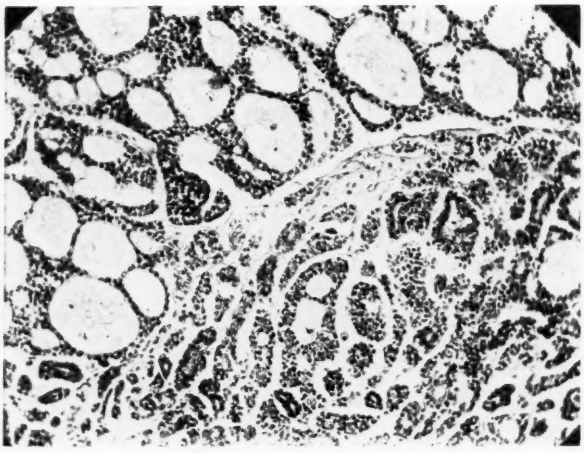

Fig. 3. Microscopic pathology of tumor in Case 3.

high and low forks could barely be heard; the Weber was lateralized to the left; the Rinne was negative four seconds, and there was no spontaneous past pointing or nystagmus. Rotation to the right and left produced nystagmus, past pointing and vertigo. The vision in the right eye was 8/10 and in the left eye 4/200. The visual field examination disclosed a nasal quadrantal anopsia and a 10 per cent constriction in the remaining quadrants. A biopsy of the mass in the nasopharynx was returned as "chronic inflammatory tissue."

He was advised to have a right temporal decompression and further x-ray therapy. The decompression was performed on November 17th, and he was discharged from the hospital. He was readmitted on February 22, 1933, complaining of dizziness, falling and further decrease in vision. At this time there was exopthalmos of the left eyeball. The visual field on the right was normal, but was markedly constricted on the left. The cranial cavity was explored by means of a left frontal bone flap, and a mass involving the base of the brain at the pituitary area was found. The mass was red in color and pulsating, and a diagnosis of aneurism of the circle of Willis was made. The mass seemed to involve the entire floor of the anterior and middle fossæ. He recovered from this operation and was not heard from until January 2, 1936, when he was admitted into the hospital seriously ill with a pneumonia of the right lung, from which he expired on January 3, 1936.

Autopsy performed on January 4, 1936, disclosed bronchopneumonia of the right lung with a fibrinous pleuritis and congestion of the left lung. There was parenchymatous degeneration of the kidneys, liver, adrenals and spleen. The heart showed brown atrophy. The most important findings were in the brain and the paranasal sinuses, the base of the brain being distorted by a lobulated tumor mass. The mass was pinkish gray, although some of the lobulations were a greenish black. It was attached to the dura at the base of the skull and extended from the crista galli in front to the sella turcica behind, and laterally on the left to the temporal portion of the parietal bone and almost to the temporal bone on the right. The

growth involved the sphenoid and ethmoid sinuses on both sides and apparently had progressed downward to invade both antra, the left more than the right. The basilar dura and the associated olfactory and optic nerves were infiltrated by the growth.

Microscopic pathology (Fig. 3) showed the tumor to be encapsulated with a fine fibrous tissue, strands of which dipped down into the rest of the mass to form the interstitial tissue. The bulk of the tumor was formed of groups of epithelial cells which were found in clusters and in acinous-like structures. The epithelial cells forming the acini have more or less ovoid nuclei. Many of the acini have a central lumen containing cellular débris and a pink staining material. The acini are surrounded by the interstitial fibrous tissue. Diagnosis: "Adenocystic basal cell carcinoma or cylindroma."

REVIEW OF THE LITERATURE

Cylindromatous basal cell growths of the respiratory passages and their adnexa are quite rare. In the search of the literature we have been able to discover but thirty-seven cases in which the clinical course, together with the histologic findings or the published photomicrographs were sufficiently characteristic to justify the diagnosis. If we add the three cases herein it would make a total of forty cases, the diagnosis of which would not be questioned, by us at least. Twenty-three, or about 58 per cent, involved the antrum. The nasal mucosa and the sphenoid sinus were each involved twice, or about 5 per cent. We were unable to gather any records of basaloma in which the ethmoids alone were involved. Most of the growths originating in the antra, however, involved the ethmoid cells sooner or later by extension. No primary growths of the frontal sinus were noted, although secondary involvement was occasionally seen. The palate was involved in one case, or about 3 per cent, although Eigler[1] reported another case. A perusal of this latter case report convinced us that the palate was involved by extension from the antrum and so it was included in the antral cases. The base of the tongue and the epiglottis were each involved in one case, or about 3 per cent. The larynx was involved about eight times, or 20 per cent of the cases. However, in two of the cases reported by Krompecher,[9] the histology was mainly that of a solid basal cell carcinoma in which some areas showed a cylindromatous architecture. The trachea was involved in but one case reported by us, or about 3 per cent of the total.

Symptomatology.—In a general way the symptomatology was dependent upon the part involved. However, there were some findings common to many of the cases. Not one of the cases reported had any involvement of the cervical lymph nodes. Distant metastases were reported in but two, or 5 per cent of the cases, the cervical ver-

tebræ being involved secondary to a growth originating in the epiglottis, reported by Eigler,[4] and the lung involved secondary to a tumor of the antrum, reported by Ribbert.[13] Recurrence after treatment by irradiation or surgery was also common. As a rule, the reappearance of the tumor was noted one to three years later, although Bagger[15] reported a case involving the antrum which showed recurrence twelve years following surgical removal. In one case of basaloma of the antrum, reported by Eigler,[4] there was a recurrence three months after combined irradiation with radium and x-ray. Yet Hofer[14] reported a case involving the antrum in which a recurrence four months after surgery disappeared after irradiation with radium and did not recur until two years later. Repeated although delayed recurrence was frequent after repeated surgery or irradiation. Growth and extension of the tumor was slow. This was evidenced by histories of nasal blockage extending over a period of one or two years, as well as the length of the time interval necessary for recurrence. Local involvement by the neoplasm was extensive. The growth invaded the parts for some distance, easily eroding cartilage and bone. The invasive characteristics of this type of growth may be better appreciated by noting the extensive involvement of the nose, palate, ethmoids, frontal sinuses, cheek and orbit found in growths originating in the antrum. In fact, Barbezet[6] reports a case in which the entire right half of the face was involved, and Ribbert[13] noted a case originating in the left antrum, which succeeded in invading the palate, antrum and lower jaw of the opposite side of the face. Our case affecting the trachea showed involvement of practically the entire length of this structure. A sessile fleshy red tumor mass with marked tendency towards bleeding was also the rule in most of the cases. In recapitulation, the findings common to most of these growths are: a sessile, red, fleshy, loosely bleeding tumor mass that shows extensive local progression, easily invading bone and cartilage, growing slowly over a relatively long period of time, with no metastases and a definite tendency towards delayed recurrence after repeated removal or irradiation.

In addition to the symptoms common to all of the growths, clinical manifestations were dependent upon the site of involvement.

Antrum.—By far the most frequent area to be involved was the maxillary sinus which occurred in 23, or 58 per cent of the 40 cases studied. The most common complaint was nasal blockage. This was present from a period of several months to a year or more. Repeated attacks of epistaxis was almost as frequent. Visual disturbances varying from impairment of vision and ocular pain to complete blindness

were noted in eight, or about 35 per cent, of the cases and was associated with orbital involvement. Headache and pain in the cheek was noted in twelve cases, or about 50 per cent. Objectively a mass in the nostril of the affected side was present in practically every case. In the earlier cases it was noted as being present in the middle meatus. In the later cases it filled the entire nasal cavity and frequently presented at the nostril and could also be seen in the postnasal space. Bulging or swelling of the cheek and the palate on the affected side were also frequent. In the cases in which visual disturbances were present a palpable mass could be found at the inner canthus, associated with exophthalmus. Transillumination and the x-ray show the obscuration of the affected sinus.

Nose.—The two cases in which the nasal cavity alone was involved evidenced symptoms of impairment of nasal respiration associated with repeated attacks of epistaxis and the presence of a mass in the nasal cavity.

Sphenoid.—The two cases of sphenoid involvement complained of headache, impairment of vision and poor hearing due, no doubt, to invasion of the eustachian tube, in addition to impaired nasal respiration, epistaxis and the presence of a mass in the nasopharynx. In basaloma of the nose and accessory sinuses death occurred most commonly from intracranial extension of the growth. This was preceded by symptoms of extreme headache and visual impairment or blindness.

Palate.—The palate was involved in most of the antral growths. One apparent case of primary growth in the palate reported by Eigler[4] complained of difficulty in swallowing for a period of two years.

Base of Tongue and Epiglottis.—One case involving the epiglottis and one involving the base of the tongue were described by Eigler.[4] Both complained of dysphagia. In the one involving the tongue there was dyspnea in addition. A reddish mass was found at the region of the foramen cecum. In the epiglottis growth hoarseness was evidenced and the mass was found to involve the right half of the epiglottis.

Larynx.—The laryngeal growths were attended with hoarseness and dyspnea, and the presence of a mass within the larynx.

Trachea.—Our case of tracheal basaloma evidenced a history of increasing dyspnea and showed bronchoscopically and roentgenologi-

cally a concentric unilateral constriction of the lumen by a firm red mass.

SUMMARY

There is a rare group of tumors found in the mucosa of the respiratory tract and its adnexa which present a typical and characteristic clinical course and unique histologic architecture. While in the past there has been some disagreement as to the histogenesis and classification of these neoplasms, it is probable that they are mucosal basal cell growths and show a comparable biologic course to basal cell tumors originating in the skin. The term cylindroma is descriptive of the peculiar morphology of these neoplasms, but they are probably more correctly termed cylindromatous basalomas. Histologically they present a picture of cell nests surrounded by a connective tissue stroma. The cell nests frequently show a central lumen containing a pink staining material and sometimes cellular débris. Clinically they are, as are their counterparts of the skin, slow growing, invasive, locally destructive, do not metastasize and recur after repeated removal. They are relatively radiosensitive and are possibly best treated by extensive resection or by adequate irradiation. Three cases, one originating in the antrum, one in the sphenoid and one in the trachea, are described and added to thirty-seven similar cases found in the literature.

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XLI.

THE "AURAL" OR "ACOUSTIC" METHOD OF TREATING DEAFNESS: FURTHER INVESTIGATION*

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The effort to stimulate or re-educate the deafened ear to increased hearing capabilities by means of sounds, usually musical, is not new but probably dates back as far as human hope has existed.

Itard,[1] in 1802, tried to stimulate the deafened ear by means of bells, drums, flutes and the human voice. Many of his followers continued the work for many years after his death. Such noted men as Alexander Graham Bell, inventor of the telephone, proposed trying the method of Itard to help the deaf to better hearing.

In 1888 Victor Urbantschitsch[1] revived the method of using the voice. This was so fatiguing to the singers that he later used sirens, tuning forks and other musical instruments but, as he thought, with less satisfactory results.

The method of Zünd-Burguet and the literature pertaining thereto was covered in a previous article[2] and will not be reviewed at this time except to state that the many favorable reports in the literature, particularly by Cathcart and Yearsley of England, have stimulated the author to continued investigation of this method of treating deafness.

The apparatus used in the present investigation is essentially an audio-oscillator of special design, which produces sound waves, claimed by the inventor to have a particular form. The audio-oscillations are secured by what is known as the beat frequency method of having two circuits which oscillate at radio frequency of approximately 200 kilocycles; the frequency of one is controlled by a variable condenser which gives an output of a slightly different frequency. This is allowed to beat against the first in a mixer tube, and the

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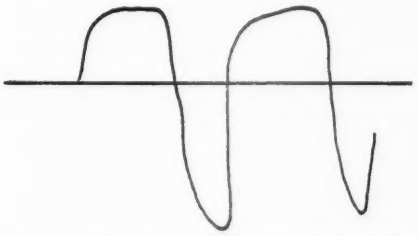

Fig. 1. The flap top wave. Frequency 1024.

difference in frequency determines the frequency of the output, which by design may be an audio-frequency.

This output is detected and fed through a resistance coupled amplifier which maintains the wave form of the output. The power output to the phones is 35 milliwatts with an amplification of 60 decibels.

The special wave form is claimed by the inventor to be what is known as a flat top (Fig. 1), as differentiated from a pure musical tone, such as is produced by a tuning fork which produces a sound wave having the form of a sine curve. This flat top wave is said to be maintained over the entire range of the apparatus, which is from 30 cycles to 10,400 cycles

The pressure pattern against the eardrum of a sound wave with a sine form would appear to be one in which the pressure would be applied and released at a steady and uniform rate, while the flat top would appear to apply the pressure quickly, hold it for a fraction of a second, release quickly and then again apply the pressure quickly, thus giving the effect of pounding in rapid successive blows.

The operation of the instrument is entirely automatic (being motor driven), except the volume control which must be adjusted to suit the individual need, so that the sound is just above the threshold of hearing. The motor is geared to drive the instrument through a complete cycle in seven and one-half minutes. That is, starting at the lowest frequency of 30 cycles up to 10,400 and back again to 30, constitutes one cycle. The advance from the low frequency to the high is made in what may be called "pilgrim steps," for the condenser which controls the frequency is advanced fifteen points and then is retarded ten points; advance is made again with the retard, and so on to the highest point, and then the same procedure followed on the return. Thus it may be said that the movement is up and down in a sort of oscillating motion. In this way the repetition of

Case No.	Patient	Age	Diagnosis	No. Treat.	Duration Treat.	Deaf for	Remarks	Change in Sensation Tinnitis	Change in Sensation Units
1	J. E.	62	Nerve type	11	6 wks.	12 years	Gradual onset	Same	+ ¾
2	F. H.	55	Ménière's sydrome	11	3 mo.	2 years	Sudden onset with vertigo	Same	−3¾
3	A. H.	30	Otosclerosis	15	6 wks.	8 years	Gradual onset	Same	+ ½
4	C. W.	46	Nerve type	21	2 wks.	7 years	Gradual onset		+2
5	E. H.	27	Nerve type	22	2 mo.	2 years			+11
6	M. D.	53	Nerve and C. C. O. M.	25	3 mo.	40 years	A. S. O. M. three weeks ago		+5¼
7	M. H.	45	Nerve type	30	9 wks.	38 years	Onset following measles		+6½
8	A. F.	45	Nerve type	30	2 mo.	6 years			−1⅓
9	L. H.	25	Otosclerosis, C. C. O. M.	36	10 wks.	Many yrs.			+1¼
10	F. J.	70	Nerve type	41	4 mo.	Many yrs.	Gradual onset		−5½
11	D. C.	42	Otosclerosis	44	2 mo.	Many yrs.		Same or worse	+1¾
12	L. R.	50	Nerve in lt. C. C. ad. O. M. in right	44	10 wks.	Many yrs.			+1
13	C. N.	58	Nerve type	48	7 wks.	Many yrs.	Gradual onset	Same	+ ½
14	K. M.	62	C. C. O. M.	52	3 mo.	Many yrs.	Hearing imp. by inf.	Same	0
15	C. K.	38	Nerve type, right	55	4 mo.	Many yrs.	Gradual onset	Same	+3½
16	A. H.	45	Nerve type	60	11 mo.	16 years	Gradual onset	Same	−2
17	J. A.	75	Nerve type	62	3 mo.	Many yrs.	Gradual onset	Worse	−7¼
18	W. F.	65	Nerve type	80	4 mo.	Many yrs.	Gradual onset		−1
19	H. N.	45	Ménière's (lt.), C. C. O. M. (rt.)	80	7 mo.	5 years	Sudden onset with vertigo and tinnitus	Same	−2½
20	C. B.	65	Nerve type	86	4 mo.	Many yrs.	Worse last year		−6
21	A. N.	71	Nerve type	86	8 mo.	Many yrs.	Gradual onset		+1¼
22	J. C.	66	Nerve type	92	4 mo.	10 years	Gradual onset. Was in much gunfire in 1896	Better	−4¾
23	M. D.	46	Otosclerosis	94	5 mo.	16 years	Gradual onset		−2½
24	H. H.	55	Nerve type	125	8 mo.	34 years	Began following pregnancy	Better	+1½
25	O. S.	55	Nerve type	148	8 mo.	Many yrs.	Gradual onset	Better	−6
26	T. C.	67	Nerve type	195	11 mo.	32 years	Gradual onset		+5
27	T. D.	56	Nerve type	204	8 mo.	Many yrs.	Gradual onset		+1

each note is secured from five to seven times with each cycle of the machine.

The clinical application of the method has consisted in giving a total of 1797 treatments to 27 partially deaf people for various types of deafness. Each treatment lasted for thirty minutes, about five times a week. Most of the patients came regularly at their scheduled time. Some were irregular with occasional lapses. Each patient had a set of ear phones and listened to the ascending and descending scale of tones. The volume was regulated by a device on each set of ear phones by each patient so as to be comfortable to his type and degree of deafness.

The ages of the patients varied from 27 to 75 years, with an average of about 52 years. They had all had impaired hearing from two to forty years, with an average hearing loss for voice of about 50 per cent. The cases were not selected but were taken as they applied to the clinic, except that no acute case was accepted.

After the diagnosis was made an audiometric examination with the 2A audiometer was done before the treatment was begun. At the conclusion of the treatment period another audiometric examination was made and the differences, if any noted. Frequent audiometric examinations were done at intervals during the treatments to check the hearing changes.

A diagnosis of nerve type deafness was made in eighteen of the twenty-seven cases. Ten of the eighteen patients with nerve type deafness showed improvement of from ¾ to 11 sensation units. Eight cases showed a loss of ¼ to 7¼ sensation units. The average of the various frequencies of 64 to 8192, inclusive of the eighteen nerve type deafness cases at the conclusion of the various treatment periods showed an increase of ¼ sensation units, a change so slight as to be without significance. The individuals in this group which showed the greatest improvement were cases Nos. 5, 7 and 26 (see chart), an increase of 11, 6½ and 5 sensation units respectively. The number of treatments were 22, 30 and 195 respectively.

Two patients (Cases Nos. 2 and 19) gave a typical history of Ménière's Syndrome. Case No. 2 was bilateral of two years' duration. A slight loss was found after eleven treatments had been given. Case No. 19 was unilateral, of five years' duration. In the latter patient a diagnosis of chronic middle ear deafness was made in the right ear. Improvement was not noted in either side after eighty treatments. A diganosis of middle ear deafness was made in five cases. Case No. 6, combined with a nerve type deafness, showed an average improve-

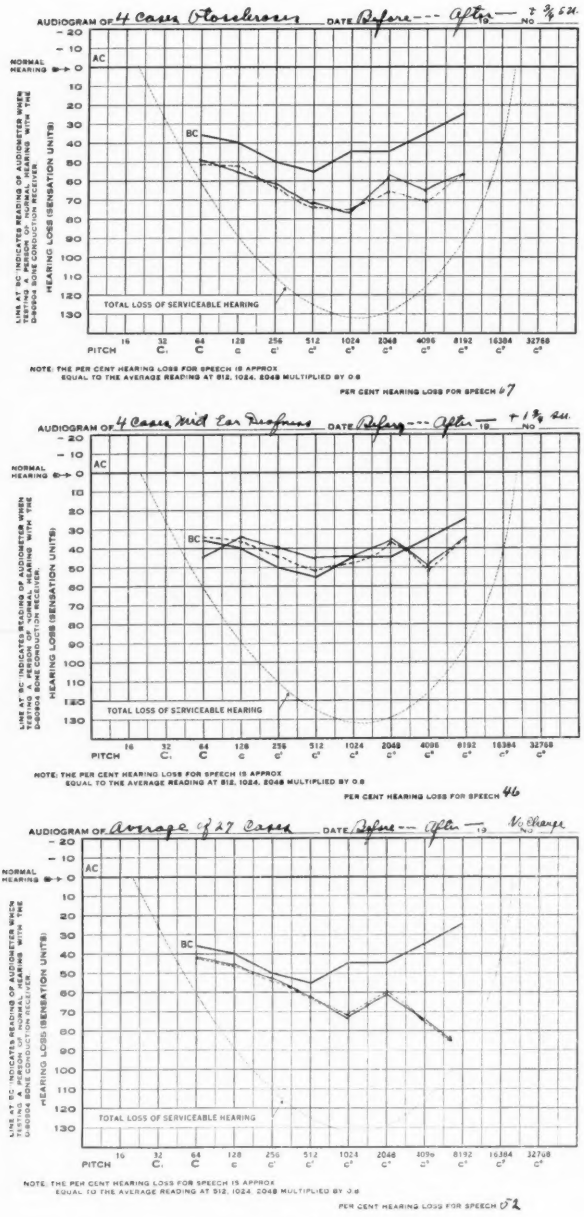
AUDIOGRAM OF 4 Cases Otosclerosis DATE Before --- After — 19 — No 3 3/4 Gn.
NORMAL HEARING
AC
BC
LINE AT BC INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE DAHOMA BONE CONDUCTION RECEIVER.
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
NOTE: THE PER CENT HEARING LOSS FOR SPEECH IS APPROX EQUAL TO THE AVERAGE READING AT 512, 1024, 2048 MULTIPLIED BY 0.8
PER CENT HEARING LOSS FOR SPEECH 67
AUDIOGRAM OF 4 Cases Mid Ear Prosfenis DATE Before --- After — 19 — No + 1 3/4 Gn.
NORMAL HEARING
AC
BC
LINE AT BC INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE DAHOMA BONE CONDUCTION RECEIVER.
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
NOTE: THE PER CENT HEARING LOSS FOR SPEECH IS APPROX EQUAL TO THE AVERAGE READING AT 512, 1024, 2048 MULTIPLIED BY 0.8
PER CENT HEARING LOSS FOR SPEECH 46
AUDIOGRAM OF Average of 27 Cases DATE Before --- After — 19 — No No Change
NORMAL HEARING
AC
BC
LINE AT BC INDICATES READING OF AUDIOMETER WHEN TESTING A PERSON OF NORMAL HEARING WITH THE DAHOMA BONE CONDUCTION RECEIVER.
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
NOTE: THE PER CENT HEARING LOSS FOR SPEECH IS APPROX EQUAL TO THE AVERAGE READING AT 512, 1024, 2048 MULTIPLIED BY 0.8
PER CENT HEARING LOSS FOR SPEECH 52

Fig. 2

ment of 5¼ sensation units. He gave a history of having had an acute suppurative otitis media three weeks before his first examination. The improvement in hearing could be attributed to the normal subsidence of the middle ear congestion. In Case No. 9, a diagnosis of middle ear deafness combined with otosclerosis was made. The improvement after thirty treatments was 1¼ sensation units, which is so small as to be without significance. Case No. 12 had a nerve type deafness in the left and a middle ear deafness in the right. The small improvement after forty-four treatments was confined to the ear with middle ear deafness. A slight loss was noted in the other ear. Case No. 14, with many years of middle ear deafness, showed no change after 52 treatments. Case No. 19 has been mentioned.

As seen from the foregoing graphs, the before and after treatment curves do not vary to any extent. In fact, in the third graph, which represents the average audiogram of twenty-seven cases taken before treatments were begun, and at the conclusion of the treatments the two graphs are almost identical.

There were four cases of otosclerosis varying from an improvement of 1¾ sensation units to a loss of 2½ sensation units, a change of not much significance, with an average improvement of ¾ sensation unit.

The average of all the frequencies from 64 to 8192 inclusive in all twenty-seven patients before treatment were begun compared with the averages at the conclusion of the various periods of treatments were identical to within one-tenth of a sensation unit. The results of the previous year's work with a somewhat different machine showed an average loss of 2¾ sensation units.

From this study it is concluded that the "aural" or "acoustic" method of treating deafness has not proved of value in the cases forming the basis of this investigation.

MARSHALL FIELD ANNEX BLDG.

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XLII.

FUNCTION OF THE STAPEDIUS MUSCLE*

ALLEN B. POTTER, M.D.

ST. LOUIS

The stapedius muscle has been the subject of considerable study. Perhaps the most valuable contribution to recent literature is an article by Doctor E. Luscher of the University of Bern. His observations were made with the use of the ear microscope, and his conclusions have been summarized as follows:

1. The tendon of the stapedius muscle was observed directly with the ear microscope with strong magnification through a non-irritated defect in a drum of an ear otherwise normal.

2. The stapedius muscle of man shows regularly and constantly appearing cochlear reflexes, which can be set up equally from the same and the opposite ear with about the same intensity.

3. With pure tones the reflex appears only in a certain tonal range. This lay, for an examined individual, between ninety and 14,000 vibrations.

4. The tone must have definite intensity, depending on the height. With rising intensity there first appears beginning jerks; finally it is a constant contraction which disappears with cessation of the tones.

5. Noises set up the reflex much more easily than pure tones. Ordinary conversation, at 50 cm. distance, likewise soft noise of the breath at the ear, light tapping of the tuning fork, etc., is sufficient to bring about a jerking. The reflex seems, therefore, especially sensitive to those stimuli which impinge themselves upon the ear in daily life. It is so sensitive that in normal daily life it must be in constant activity.

6. The stronger, higher and noisier the sound impression becomes, the more easily the reflex appears.

7. The setting up of the reflex is caused by mere anticipation of a strong sound stimulus. However, the expectation to a certain

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degree must be restricted reflexively, and cannot be brought about through listening.

8. The reflex can be released through slight stimulation; for an example, through pricking of the external ear.

9. Even with the strongest voluntary contraction of the muscles innervated by the facial nerve no simultaneous contraction of the stapedius occurs. A voluntary contraction of the stapedius cannot be established.

10. The contraction of the stapedius seems to be isometric and no movement of the drum seems to follow.

11. All facts point to the conclusion that the contraction of the stapedius muscle causes a decrease of the sound transmission and thereby a decrease in hearing. From this viewpoint it is to be looked upon not as an agency for hearing but as a protective arrangement which has the function in a reflective automatic from strong sound effects. The corresponding animal experiments of Kato were confirmed in the above description of the human ear examination.

I have repeatedly studied structures of the middle ear and their functions through large perforations, many of which presented very little pathology either of the ear drum—except for the perforation—or of the middle ear. With the use of the ear microscope any part of the ear drum may be brought under observation with a magnification ranging from five to fifty times.

The most valuable condition for study that has come under my observation was an ear drum with a large traumatic perforation of the posterior superior quadrant. I had examined the ears before, and knew the hearing in both ears to be within normal limits. The membrana tympani was not thickened and no discharge was present. The stapedius muscle was seen free in the tympanic cavity with its attachment to the posterior wall and the stapes intact. The fine blood vessels could be seen coursing along the tendon. This was an ideal condition to use as a control on other observations where pathology had been present. In the most favorable cases for observation the audiogram and the bone conductions were made for both ears.

We might say here that it is an accepted fact that the contraction of the stapedius is the same for the two ears. That is, in the ear stimulated and in the opposite ear the contractions are the same.

In the beginning of the observations, I first used the Galton whistle to determine the maximum contraction of the stapedius, to be assured that there was no mechanical interference with motion.

Then with the use of the audiometer the contractions of the stapedius were tested. The ear phone was placed on the opposite ear. The intensity of the different notes was increased until the contraction was first noticed. I attempted to obtain a curve representing the different notes and the minimum intensity at which a contraction was noted. The curves represent those obtained with the use of the audiometer, and it is quite logical that this curve would be found throughout the perceptible tone range if the intensity of the lower and higher notes were increased. It was found that this curve followed closely the one for bone conduction but placed at a lower level.

It is interesting to note that these contractions are not made up of a series, but one continuous contraction of the stapedius with a very short latent period. The period of relaxation is apparently equal to that of contraction. Let us take an example. When sounding a note of given intensity there is a contraction of the stapedius muscle. The contraction continues during the stimulation—if the intensity is increased the contraction increases, and so on up to the maximum contraction. Upon cessation of the stimulus the relaxation takes place immediately. The stapedius muscle is not easily fatigued. It is in a state of relaxation under ordinary conditions.

It might be expected that if the function of the stapedius is purely protective that some arrangement would be made for an anticipated over-stimulus, and the eye seems to be a logical source from which to receive such a reflex or stimulation. It is known that if one end of a rubber tubing is placed in the external canal of an ear, the other end between the lips, and the individual, while gazing fixedly at some object, utters a sharp quick sound, the light will be seen suddenly to move at an angle of forty-five toward the stimulated ear. With two tubes similarly placed, one in either ear, no movement results. In the light of what is known about the cause of nystagmus, one infers that the nystagmoid movement was caused by the sudden increase in vestibular pressure, which, however, was compensated for by an adjustment of the mechanism, an adjustment which remains constant so long as the stimulus remains unchanged in volume and pitch. It might be explained thus: The ear is adjusted to a relatively weak intensity of sound. The arrangement provides for all possible amplification of the incoming sound. While the ear is so adjusted the first few notes of a sudden intense sound piles over into the unprotected sensory organs of the ampullæ, thus producing the nystagmus and also stimulating the sensory endings in the vestibule and thus probably furnishing the afferent impulses involved in the reflex compensation of the muscles of the middle ear.

The foregoing experiment was mentioned to show the close relationship of the ear and eye. We are as yet not satisfied with our explanation of this interrelation, but do know that it exists.

I shall try to show that the stimulation of the stapedius muscle does not depend entirely upon the stimulation of the organ of Corti, which in turn sends impulses to this protective mechanism, but that the contraction may be brought about by mere anticipation and that this anticipation may be caused by the association of loud noises with the stimulation of the eye or other sensory organs.

This experiment was carried out in a soundproof room. The stapedius muscle showed no contractions under fifty times magnification before the experiment was started. Then an ordinary repeating cap pistol was snapped several times without caps, and at a distance of three feet from the patient's ear. There followed no contraction of the stapedius. The pistol was then loaded; at each explosion of a cap there was a maximum contraction of the stapedius muscle, then there came a few snaps of the pistol without the cap explosion and no contraction was noted. The pistol was then handed to the patient, who was instructed to pull the trigger at the rate of about thirty per minute. At each explosion there was the expected contraction; then came several intervening blanks and at each click there was also noted strong contraction. Again a series of explosions followed by a series of blanks. This experiment was repeated at different times, and with three different individuals. All showed conclusively that the stapedius muscle does function as a protective mechanism, not altogether as a cochlear reflex, but also through anticipation of an overstimulus of the organ of Corti.

Again let us take an unusual case which has been carefully studied. The left membrana tympani had a large perforation permitting an excellent view of the long process of the incus and stapedius muscle. While stimulating the right ear the action of the stapedius was watched through the ear microscope, thirty times magnification. The response was the same as shown in the previous experiment. Audiograms of both ears were made for record. The right membrana tympani was quite retracted and gave the appearance of almost complete adherence to the inner wall of the middle ear. The long process of the incus was seen to be attached to the membrana tympani similar to the long process of the malleus, even to the extent of having the blood vessels coursing down along its anterior and posterior border. The stapedius muscle could be seen through the thin retracted membrane. There were no rhythmic contractions, but it did

respond to stimulation of the opposite ear, and with practically the same intensity. I mention this particular case first because the observations were carried out through a retracted but otherwise normal drum, and second because of the excellent hearing which was present.

The contraction of the stapedius could not in this case alter the tension of the ossicular chain, which seems to be of minor importance so far as acuity in hearing is concerned. On the other hand, it is most logical that the excellent hearing in this case was dependent upon the fact that there was free excursion of the stapes in the oval window and unimpaired function of the stapedius muscle.

Complete relaxation of the stapedius is necessary in order to get the greatest acuity in hearing. Fibrous bands or a shortening of the stapedius muscle could act in the same capacity as contraction of the muscle, under which condition the acuity of hearing is decreased.

It is well here to mention the effect of our masking apparatus which we use while doing our hearing tests, whether this be by use of the audiometer or spoken and whispered voice. If we mask with a note of sufficient intensity to cause a contraction of the stapedius muscle we have cut down on the acuity of hearing in the ear examined. In other words, from my charts I would suggest that the masking be made by an intensity less than that which shows contraction of the stapedius. This varies for the different notes throughout the perceptible tone range. It is also known that high tones will mask low tones, but low tones will not mask the high tones.

Let me mention a condition which has frequently come under our observation. The patient complains of loud noises or even of conversational voice producing annoying rasping sounds in the affected ear. This may be during an acute, subacute or chronic stage. Never is this annoyance caused by tones which are not sufficient to stimulate a contraction of the stapedius muscle. And these noises are more apt to be caused by tones lying between 128 and 1024 double vibrations. Until it is proved otherwise, I shall be inclined to consider this condition as a temporary paralysis or paresis of the stapedius muscle, under which condition it is unable to carry out its protective function and act as a soft pedal to the organ of Corti.

I offer this paper not because of my present findings, but because it does present a new angle for considering some of our important problems in otolaryngology.

CONCLUSIONS.

1. The function of the stapedius muscle is purely protective.

2. Contraction of the stapedius may be brought about by

   a) Reflex from the organ of Corti or cochlear reflex.

   b) Anticipation of loud sound.

   c) Mechanical stimulation of external canal.

3. Contraction is the same in both ears.

4. The graphic representation of the contraction of the stapedius muscle for different notes is dependent upon the acuity of hearing for these notes and is found to be similar to the audiogram but lying at a lower level—30 to 40 units intensity.

5. We should select more carefully the pitch and intensity used for masking the opposite ear.

6. We are continually dealing with conditions due to impaired function of the stapedius muscle.

UNIVERSITY CLUB BUILDING.

XLIII.

ENDOSCOPY FOR FOREIGN BODY: REPORT OF 178 CASES OF FOREIGN BODY IN THE AIR AND FOOD PASSAGES*

CHEVALIER LAWRENCE JACKSON, M.D.

PHILADELPHIA

The details of the individual cases in this series will be published in tables.[1] Space will permit me only to give a statistical summary of the series and to discuss briefly some of the various groups of cases. The largest group was that constituted by the cases in which bones were the offending objects, this group numbering thirty-eight. The next most numerous group was that of safety-pins, of which there were twenty-one. Third came peanuts, numbering eighteen, though if we group the peanut cases with the other cases of nuts, seeds and shells, into a group of vegetable foreign bodies, this composite group would tie the bones for first place. Among the nuts other than peanuts there were chestnuts, Brazil nuts and pecans. The next most numerous group were coins, with seventeen cases, and meat with fifteen cases. Screws numbered seven; common pins five; while nails, orange seeds, watermelon seeds, buttons, beads, tacks and jackstones numbered three each. Beans, collar buttons, prune-pits, plum-pits, grains of corn, cartridge shells and eggshells numbered two cases each, and there were twenty objects of which there was one each: pea, tooth-paste cap, key can opener, ringbinder, screw eye, piece of wire, tooth, tooth-plate, broncholith, crust of secretion, chestnut shell, raisin, pumpkin seed, religious medal, metal disc, rivet, tin foil, bobbette pin, portion of buckle, and wrist watch.

VEGETABLE FOREIGN BODIES.—Let us consider first of all the large and important group of vegetable foreign bodies. The diagnosis of this sort of case has been fully discussed elsewhere,[1 2 3] and the characteristic signs of obstructive atelectasis and obstructive emphysema have been carefully described. In connection with the diagnosis of vegetable "nonopaque" foreign bodies, therefore, I wish to emphasize only one or two facts. Though a great deal has been said about the importance of obstructive emphysema of one lung or of one lobe,

*Read before the Section on Eye, Ear, Nose and Throat Diseases of the Medical Society of the State of Pennsylvania, Harrisburg Session, October 3, 1935.

Fig. 1. Pneumothorax of left lung following attempt at thoracentesis in child with perfectly definite history of foreign body. Obstructive atelectasis is often mistaken for empyema or pleural effusion, as it was in this case, but in a patient with a foreign body history, diagnostic bronchoscopy should be done before needling. Bronchoscopic removal of the fragment of peanut was postponed until after re-expansion of the lung.

little has been said about the importance of bilateral emphysema. This condition is quite often encountered in cases of nonopaque foreign body, and I feel that it should be emphasized, since it is very easily overlooked. Not only the relative movement of the diaphragms should be checked in the fluoroscopic examination, but also the degree of movement. If there is some bilateral restriction, and especially if there is, in addition, a wheeze heard at the open mouth, a tentative diagnosis of foreign body is justifiable and bronchoscopy indicated. The foreign body may be in the trachea, or there may be two fragments, one in each main bronchus.

Another diagnostic point worthy of emphasis is the importance of recognizing obstructive atelectasis and not mistaking it for empyema. Certainly if there is any foreign body history, have bronchoscopy done before needling. In one of my cases, a child with a perfectly definite foreign body history had developed a pneumothorax following negative thoracentesis, and we had to wait several weeks before doing bronchoscopy for removal of the peanut (Fig. 1). One

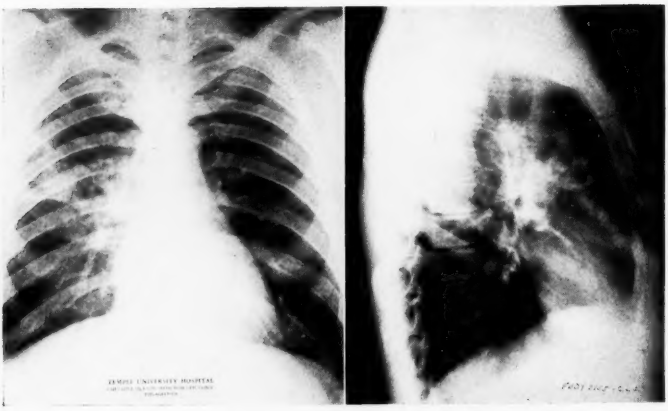

Fig. 2. Pneumonitis of right upper lobe in adult male with peanut kernel lodged in right upper lobe bronchus for seven months.

of the peanuts was lodged in the esophagus of a young woman with esophageal stenosis due to carcinoma, and one had been in the right upper lobe bronchial orifice of an adult male for seven months (Fig. 2); the others were all in the bronchi of children. Two of the watermelon seeds were lodged in the trachea and gave very little in the way of physical or roentgen-ray signs; one was in a bronchus, and three previous attempts at removal had been made elsewhere; the fourth was in a stenosed esophagus. Of the three cases of orange seed, two were lodged in old cicatricial stenoses of the esophagus and one in a cancerous stricture. The two plum-pits and the two prune-pits were likewise lodged in esophageal strictures. One of the bean cases was one of the two fatal cases in the series. The child had suffered prolonged cyanosis due to complete obstruction of the right bronchus, with atelectasis of the right lung and partial obstruction of the left bronchus. Despite the fact that the foreign body was removed without difficulty, this child died about seven hours later. In the other fatal case, the foreign body was a peanut and death was attributed to the very severe acute inflammatory reaction resulting from the previous use of ether, which greatly aggravated the "arachidic bronchitis" already present.

Tracheotomy was required in four of the peanut cases, one of the bean cases, one of the jackstone cases, one of the screw cases and the case of chestnut shell. It had been done previous to admission in one of the nut kernel cases, and in one of the safety pin cases an

Fig. 3. Bone in the cervical esophagus. Note ossification of laryngeal and tracheal cartilages. Not infrequently spots of ossification are mistaken for foreign bodies.

intubation tube had been inserted following an unsuccessful attempt at removal. In this last case we replaced the intubation tube with a tracheotomy before bronchoscopy for removal of the foreign body. In all cases the foreign body was removed through the mouth. Unquestionably the use of the oxygen tent in some of the later cases has obviated tracheotomy, and it should be used routinely in the postoperative care of patients in whom subglottic edema is anticipated. We have substituted it for the "croup tent" formerly used in these cases.

BONES.—Three of the bone cases were bronchial and two of the three have been reported previously.[4] In one of these cases the x-ray showed partial atelectasis of the obstructed lobe, in another it was entirely negative, and in the third it showed a typical obstructive emphysema.

Bones in the esophagus are generally well visualized in properly taken neck films, even without the use of an opaque mixture (Fig. 3), but sometimes their differential diagnosis from ossification of the

Fig. 4. Safety pin in right lower lobe bronchus of a child four and one-half years of age. Note that the foreign body has remained in the sagittal plane, that in which all foreign bodies pass through the glottis.

laryngeal cartilages requires careful study, as has been shown by Chamberlain and Young.[5] In one of the cases of bone in the esophagus the patient developed a retroesophageal abscess a week after the removal of the bone. This abscess was successfully drained by cervical mediastinotomy and the patient had an uneventful and rapid convalescence. There are some cases in which the ultra-conservative treatment of such complications is probably indicated, and others in which an abscess may be opened through the esophageal speculum or the esophagoscope; but cervical mediastinotomy was fully justified in this case by the excellent result obtained.

Safety Pins.—All of the safety pins but one were open. Ten were in a bronchus (Fig. 4), eight in the hypopharynx or esophagus, and three in the larynx. Previous attempts at removal had been made in three of these cases, but all the patients were in good condition. It is interesting to note that all but three of the patients with safety pins were children, and eight were under one year of age.

Coins.—All of the coins were lodged in the cervical esophagus, except a dime that was lodged above a congenital stricture at the junction of a congenitally short esophagus and thoracic stomach in a child, and a penny which was lodged in the right main bronchus of a man. The latter patient had been amusing his children by toss-

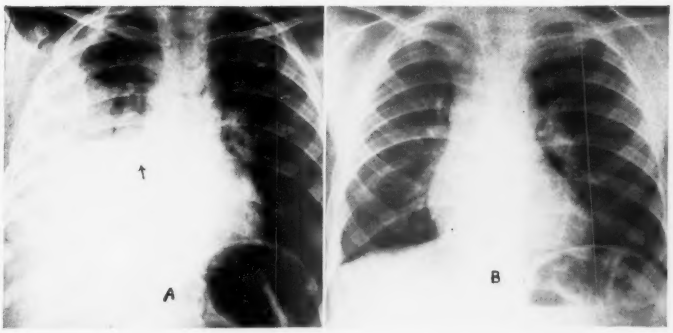

Fig. 5. (*A*) Tack lodged in right bronchus for almost two years. Bronchoscopic removal was accomplished in a few minutes, but immediate subsequent surgical drainage of the associated empyema was advised. This case illustrates the role that bronchial obstruction can play in the etiology of empyema. Bronchoscopy should be done to exclude bronchial obstruction by nonopaque foreign body or bronchial stenosis in all cases of empyema.

(*B*) Film taken about six months later, showing complete restoration to normalcy. Complete recovery followed bronchoscopic removal of the foreign body and surgical drainage of the empyema. (W. E. Burnett.) Bronchography showed a very slight bronchiectasis in the right lower lobe, but the child was symptom-free.

ing coins up into the air and catching them in his mouth. One other coin case was of especial interest because the child had been sent away without an x-ray examination by the interne on duty in the accident dispensary of one of the large Philadelphia hospitals, and by two different physicians, with the assurance that if a coin had been swallowed, it would surely pass! Finally, ten days after the accident, this boy's father took him to another hospital and insisted on x-ray examination, which showed the coin.

Screws and Nails.—Of the seven screws, three were lodged head up and their removal had been unsuccessfully attempted elsewhere. One of the nails was likewise lodged head upward and its removal had been unsuccessfully attempted. The difficulty in these cases generally lies in getting the forceps blades to displace the bronchial wall sufficiently to permit them to slide down over the head of the screw or nail. Stiff expanding forceps are required, and sometimes the use of an expanding bronchoscope is of aid in dilating the bronchus proximal to the foreign body. The guidance of the double plane fluoroscope is a great advantage in dealing with cases of this sort.

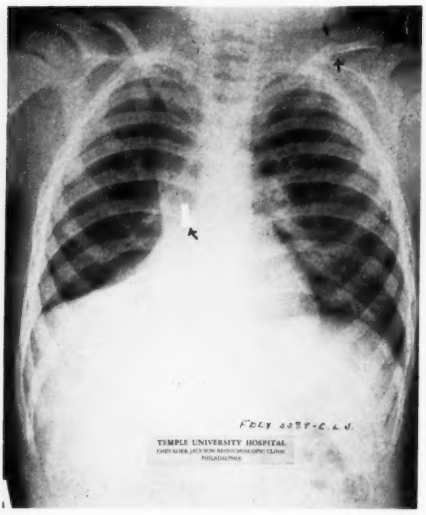

Fig. 6. Rivet lodged in right bronchus discovered accidentally when patient was being x-rayed for study of a fracture of the left clavicle.

Opaque Foreign Bodies Not Diagnosed Because of Failure to Take X-Ray.—The overlooked coin in the esophagus has already been mentioned. Another child was treated for many months for "asthma" because he had a wheeze, yet the cause of the wheeze was a large screw lodged in the right bronchus. Another child was treated for tuberculosis because no x-ray examination had been made, and the carpet tack in his bronchial tree had never been discovered. In one of the most recent cases a tack had been lodged in a little boy's right lower lobe bronchus for almost two years, and he had developed a pulmonary abscess and empyema, just because the x-ray taken at the time of the accident included only the abdomen. (Fig. 5). The tack was easily removed with the bronchoscope, but the empyema required surgical drainage. We have frequently called attention to the fact that in cases of suspected foreign body, x-ray examination should include the entire area from the nasopharynx to the tuberosities of the ischia. In some of the cases of overlooked foreign body there has been absolutely no history of the accident and it is discovered incidentally when x-ray examination is made for some other reason, as in the case of a boy who was having x-ray study of a fractured clavicle when a rivet was discovered in his right bronchus (Fig. 6). This foreign body had apparently been present for at

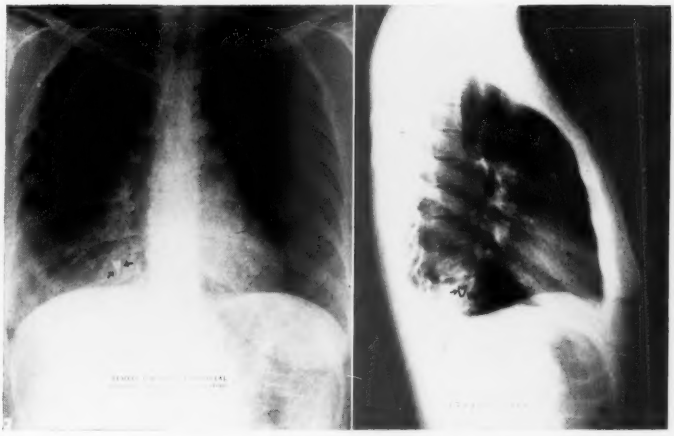

Fig. 7. Tooth in branch of right lower lobe bronchus, removed bronchoscopically without difficulty, under biplane fluoroscopic guidance.

least several months, because there was a rather marked degree of chronic inflammatory reaction about it.

Fluoroscopic Aid.—The aid of the biplane fluoroscope was used to advantage in a number of the cases in the series. Fluoroscopic aid is indicated in practically all cases of safety pins, and in all cases of opaque bronchial foreign bodies which have a tendency to work out into the more peripheral parts of the bronchial tree, such as common pins[6] (Figs. 7 and 8). It is also necessary in the removal of foreign bodies from the stomach.[7] The fluoroscope should be biplane so that the patient does not need to be turned, and position should be checked repeatedly, first in one plane, then in the other, in all manipulations. Even when working with fluoroscopic guidance, however, one should make as much use of direct vision through the tube as possible. Very often endoscopic inspection will explain and solve problems encountered when working under fluoroscopic guidance. The fundamental rule in advancing bronchoscope and forceps to contact with a foreign body at the periphery is to "find the lumen," and when difficulty is experienced in doing this, direct inspection may reveal the orifice of the desired branch bronchus.

Dr. W. Edward Chamberlain has designed a biplane fluoroscope for our clinic at the Temple University Hospital which has a number of distinct advantages.[2,3,8] The tube which supplies the horizontal ray is mounted behind an aluminum wall at a distance of four feet

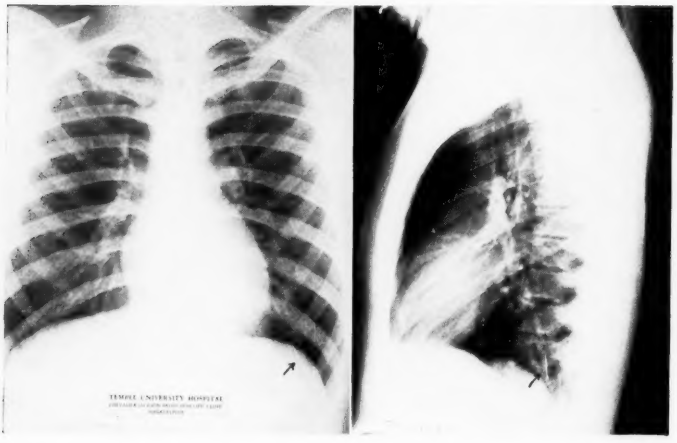

Fig. 8. Common pin in peripheral branch of left lower lobe bronchus. This foreign body was removed by costophrenic bronchoscopy with biplane fluoroscopic guidance.

from the patient, allowing ample room for the head-holding assistant, while at the same time giving less distortion, and less exposure of the patient's skin. The tube supplying the vertical ray is mounted beneath the floor. The fluorescent screen is mounted on the same framework that supports the tubes, so that it is always properly centered. Another feature of the apparatus is a pair of electric clocks which automatically record the time of exposure to the horizontal and the vertical ray respectively. Since the beam given off by both tubes has been accurately calibrated by means of the ionization chamber, it is possible to estimate exactly the skin exposure the patient is receiving.

CONCLUSIONS

1. In a series of 178 consecutive cases of foreign body in the air and food passages, bones, safety pins, peanuts and coins were the most numerous offenders, in the order named; and if the peanuts were grouped together with other nuts, seeds and shells, the composite vegetable group would tie the bones for first place.

2. Bilateral emphysema is an important finding, suggestive of tracheal or bilateral bronchial obstruction, and constitutes an urgent indication for bronchoscopy, especially if associated with a wheeze in a patient with a history of foreign body.

3. The differential diagnosis of obstructive atelectasis and empyema is sometimes difficult, but in a patient with a foreign body history, do bronchoscopy before needling.

4. There were two fatal cases in this series, one a case of bean with atelectasis of one lung and partial obstruction of the opposite bronchus, in which prolonged anoxemia had caused fatal damage to the medullary centers; and the other a case of peanut in which ether had been used as a general anesthetic in a previous unsuccessful attempt at removal.

5. Tracheotomy was required postoperatively for dyspnea due to subglottic edema in six of the twenty-five cases of vegetable foreign body in which the foreign body was lodged in the tracheobronchial tree. It was also required in a case of jackstone in the hypopharynx and upper esophagus and in a case of screw in the bronchus. It had been done by our predecessor in one of the bead cases, and in one of the cases of nut kernel. One of the patients with a safety pin in the bronchus had an intubation tube in place on admission, which we replaced with a tracheotomy tube. Unquestionably the oxygen tent offers considerable promise of obviating tracheotomy in some of the cases which would otherwise require it, and the oxygen tent is to be preferred to the "croup tent" in the postoperative care of bronchoscopic cases in which subglottic edema is present or anticipated.

6. Bones in the esophagus are generally visualized in good neck films, even without the use of an opaque mixture, but it is important to differentiate them from localized spots of ossification in the laryngeal cartilages.

7. Cervical mediastinotomy may be a very satsifactory procedure in certain cases of suppurative complications of bone in the esophagus.

8. Safety pins were found to be about equally divided between the air and food passages, with twelve in the former and ten in the latter.

9. In twenty-two of the cases in the entire series unsuccessful attempts at removal had been made elsewhere, but in only one case had serious harm been done at the previous endoscopy, and in that case the harm was not done by instrumentation but by the administration of ether in the presence of an "arachidic bronchitis."

10. In several of the cases opaque foreign bodies had been overlooked for considerable periods of time because the physician hesi-

tated to have roentgen ray examination made, with the result that the patient was treated for asthma, tuberculosis or some other condition.

11. Biplane fluoroscopic aid is invaluable in cases of safety pin and many other opaque foreign bodies, especially those like nails, screws and common pins, which have a tendency to work further and further out into the peripheral bronchi. The ideal apparatus among those thus far developed is thought to be the one above described, in which "the telefluoroscopic feature reduces distortion, increases detail and produces a reduction in the degree of x-ray exposure of the patient's skin for identical illumination," at the same time keeping the horizontal ray tube far out of the way of the bronchoscopist's head-holding assistant.

3432 NORTH BROAD ST.

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XLIV.

THE EARLY HISTORY OF OTOLARYNGOLOGY IN AMERICA WITH SPECIAL REFERENCE TO THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY*

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NEW YORK

The story of the beginnings of laryngology and of otology in this country well antedates the epoch making discovery, in 1855, of the laryngoscope. In 1821, William Pearce, a surgeon to the Pennsylvania Hospital, brought out an American edition of the "Anatomy of the Human Ear," by John Cunningham Saunders, an Englishman. A few years later, Nathan R. Smith, a distinguished surgeon of Baltimore, translated from the French, Saissy's book on the "Diseases of the Internal Ear" and wrote a chapter in it on the external ear, calling attention to the use and value of the catheter and recommended the method of Kramer of examining the drum membrane by direct sunlight, while in 1846, a Baltimore physician by the name of Dr. Joshua L. Cohen, with a Dr. Dix of Boston, became distinguished for the particular attention he paid in his practice to the internal ear and to the investigation of abnormal conditions of the tympanum and eustachian tube. Reeves' American edition of Cooper's "Dictionary of Practical Surgery," 1846, says of them: "In cases of deafness, these gentlemen have employed a condensing apparatus for administering the air douche through the eustachian tube, after the plan of Kramer and others. By the air and also by the water douche, these gentlemen have acquired great tact in the diagnosis and treatment of obstructions in the tube and upon the tympanum." This testifies to an actual knowledge of the anatomy of the ear and of the treatment of affections of the sound conduction apparatus at a time well in advance of the birth of the specialty of otolaryngology. This birth actually took place in 1855.

To Garcia, a Spaniard, living in England, is accorded the high honor of being the discoverer of the laryngoscope. Türck and

*Presented before the American Laryngological, Rhinological and Otological Society, Denver, May 18, 1936.

Czermak, of different nationalities but both living in Vienna, at the same time, contended for the credit of first employing it in medicine.

What share was our own country taking in these strivings and seekings while still "rosy-fingered dawn" filled the otolaryngologic sky. To the early members of this society there was no need of rehearsing this tale. Since their day, however, another generation of laryngologists and otologists, mighty in number and boasting in the strength of their youth, has arisen, to whom this story is a dim tradition or an unread tale. Let it be said, then, that America's share in this development is one which can be pointed to with just pride. Keen minds on this side the water were pondering over the same problems which confronted Türck and Czermak. Living in Woburn, Mass., at this time was a young physician by the name of Ephraim Cutter, a graduate of Harvard and of the University of Pennsylvania, who busied himself with a solution of the problem how to see the larynx by means of prisms. After much study he submitted his ideas to G. B. Clark of the firm of Alvan Clark and Sons, the celebrated lens makers of Cambridge, Mass., with the request that he construct an instrument according to the drawing. This, with certain necessary modifications, Clark did in the year 1857, as is shown by the correspondence which passed between them and which was later published by Elsberg. Cutter afterward moved to New York City, where he was still living in 1906. While this arrangement of mirrors was, as far as we can learn, never actually used, it is of no little interest to know that such work had been done by an independent American mind.

Cutter's work was theoretic. For many years antedating him the true pioneer in laryngology in this country had been daily treating diseases of the larynx by the same measures we employ today. The name of Horace Green will ever stand as the father of American laryngology. Few men in the medical profession have been more bitterly assailed. He, like many another pioneer in the fields of truth, was in advance of his generation, and ridicule and calumny were his lot. More fortunate than the majority of that class, it was his happy fate to live to see his claims vindicated.

Green was born on December 24, 1802, and died November 29, 1866. After graduating in medicine, he settled in Rutland, Vt. In 1835 he moved to the city of New York. He was one of the founders of the New York Medical College, of which he was president and where for a long time he occupied the chair of medicine. He received many honorary degrees, including that of LL.D. from the University of Vermont. He continued to practice his specialty till shortly before

his death. His biography speaks of his being endowed with a strong religious sentiment. In 1846 Green brought out his treatise on "Diseases of the Air Passages." In this he claimed to cure diseases of the larynx by direct applications of varying strengths of nitrate of silver, introduced upon a sponge with a curved handle. He states that he first did this in 1838, shortly after his return from a year's visit in Europe. The idea of employing direct medication he ascribes to a conversation with an English physician whom he met while abroad, and he disclaims adopting it from Trousseau or Belloc. His was the first treatise on laryngology published in America.

The publication of Green's book provoked intense controversy. As he says, for many years he refrained on this account from again asserting his views. In 1857, however, he once more set them forth in a paper read before the New York Academy of Medicine. In this paper he claimed to be able not only to introduce instruments into the larynx but also into the bronchi, and asked that a committee from the Academy be appointed to invsetigate his claim. This was done, and under the chairmanship of the celebrated Valentine Mott, it reported that while the latter part of his contention lacked sufficient proof, in the main his claims were just.

While Green stands forth as the one conspicuous laryngologist of the first half century, others of his countrymen had shown their interest in this special field of science during the prelaryngoscopic period. Cheesman, in 1817, described in detail a laryngeal growth. In 1828 Physick of Philadelphia published a description of the tonsillotome which bears his name. Before that time it was customary to put a ligature upon the tonsils and allow it to remain for several days. In 1832, Fahnstock of Lancaster, Pa., described one which closely resembles the Mathieu tonsillotome, long in use when it was the procedure to remove only part of the tonsil.

Acute and chronic laryngitis, which are wont to present no symptoms of gravity, were to the men of that day regarded as among the most deadly of diseases. The literature of those years is full of reports of a suffocative form of laryngitis, usually fatal, which is rarely met today. In its chronic form it was held to be almost invariably tubercular in origin. Green published, in 1858, the fourth edition of his work, as well as in 1852, one on the "Treatment of Polyps in the Larynx." Here he showed a number of instruments and reports and several cures by intralaryngeal removal.

In 1858, Czermak published his first paper on the laryngoscope. Two years later Dr. Hugh Strangenwald read a paper before the New

York Medicochirurgical Society describing it. March 6, 1861, Dr. William H. Church presented the subject at an interesting meeting of the New York Academy of Medicine. On that occasion Dr. Ernest Krackswizer asserted that in all probability he was the first person to use the laryngoscope in America. He used a reflector, devised by Semeleder, an assistant of Czermak. Among others, Horace Green was present and reported his experiences with the new instrument. What a memorable evening for that battle-scarred old warrior! How proud he must have felt to hear his contentions of a lifetime, after meeting allegations of the gravest character, fully justified!

Garcia's memorable discovery aroused profound interest in the subject of laryngology and otology throughout the entire world. In this interest America was not lacking. In a scholarly address, delivered in 1929, on the occasion of the fiftieth anniversary of the American Laryngological Association, Dr. D. Bryson Delavan paid just tribute to the share that a group of recent medical graduates had in the development of the specialty of laryngology in this country. Among the most prominent of these was Louis Elsberg of New York, who was one of the first in America to give instructions in laryngology, being preceded only by J. Solis-Cohen of Philadelphia, who in 1866 instituted regularly organized courses in laryngology at the Philadelphia School of Anatomy, and by Dr. H. K. Oliver very shortly after at the Harvard Medical School. Solis-Cohen, in 1872, brought out the first book on "Diseases of the Throat," following the advent of the laryngoscope in America. Still another was Clinton Wagner, "who, in 1873, established in New York City the Metropolitan Throat Hospital, the first hospital in this country exclusively devoted to the study and treatment of diseases of the nose and throat, and inaugurated in it the first regularly organized courses of instruction for graduates in medicine." In this immortal company belongs Lefferts, for a long time professor of laryngology in the College of Physicians and Surgeons, and a little later appeared Bosworth, to whom is due the credit of first stressing the importance of correction of nasal obstructions.

The interest that these men in particular took in this new specialty led to the organization, in 1873, of the New York Laryngological Society, the first of its kind in the world, which continued until it was merged, in 1886, with the Section of Laryngology of the New York Academy of Medicine. The enthusiasm and scientific interest in the subject which possessed the little group in New York spread throughout the country and led to the organizing in Buffalo, in 1878, of the American Laryngological Association, which is famed

today for the outstanding work that it has given rise to for now fifty-eight years.

At the time we are speaking of, and for a long time afterward, the specialty of otology was generally regarded as the twin brother of ophthalmology and remained so until very recently. At the same time that Elsberg, Wagner and Solis-Cohen were making their imperishable contributions to laryngology, another group of men was aroused by Garcia's discovery to devote themselves to the study of the ear. Of these the most conspicuous were D. B. St. John Roosa of New York, Clarence J. Blake of Boston, Albert H. Buck and Samuel Sexton of New York, J. O. Green of Boston and later Herman Knapp of New York. Most of these had studied with Politzer in Vienna and had returned to this country in the late sixties and early seventies eager to carry on the work they had been inspired to begin while students abroad. Roosa translated, in 1864, Von Tröltsch's "Diseases of the Ear," and in 1873 brought out his own authoritative work on "Diseases of the Ear," in the preface of which he points out the submerged position that otology had occupied and expressed the opinion that "The day will soon arrive—if indeed it be not already upon us—when otology will take equal rank with ophthalmology, to which department it has so long been a mere appendage, and when some knowledge of the diseases and treatment of the ear will be required of every practitioner."

The American Ophthalmological Society was founded in 1864. At first it was thought that otologic subjects could be considered simultaneously with those having to do with ophthalmology at the meetings of the Society, but it was soon found not feasible, and in 1868 the American Otological Society was founded, the first society of its kind in the world. The American Laryngological Association followed it ten years later. The American Otological Society and the American Laryngological Association, since their establishment, have served as the forum for the presentation of many important contributions on laryngology and otology. Both societies were established when the number of those devoting themselves to these specialties was small. In the case of one of them the membership was strictly limited by its constitution and in the other by general agreement. It required, however, only a few years for the specialties to grow, and in a short time a considerable group of young men, many of whom had received their training in Europe, had established themselves in this country. The limitations to membership in the two national societies made it impossible for most of these to become members. The need of proper society connections became more and

more apparent, and after long and earnest consideration a group of those most interested came together by appointment, on the 19th day of June, 1895, at 8:30 P. M., at 25 West Thirty-sixth street, New York City, and organized a Society which was named The American Laryngological, Rhinological and Otological Society, and elected Edward Bradford Dench as president; Joseph O'Dwyer, vice-president, and Robert C. Myles as secretary-treasurer. There were eleven men present at this eventful organization meeting—T. Passmore Berens, Lewis A. Coffin, H. H. Curtis, Dwight L. Hubbard, Robert C. Myles, Wendell C. Phillips, J. W. Brennan, Henry Coggeshall, W. L. Culbert, N. J. Hepburn and Frank E. Miller and acceptances were received from G. E. Brewer, Brentano Clemens, L. A. Dessar, E. B. Dench, Richard Frothingham, T. J. Harris, Joseph O'Dwyer, F. J. Quinlan, Frank W. Ring, J. L. Adams and Dillon Brown. At this meeting a committee on Constitution and By-laws was appointed, consisting of Drs. Berens, Curtis, Hubbard, Phillips and Coggeshall. At the second annual meeting in New York, April 17, 1896, a Constitution and By-laws were adopted. The society for the first year functioned under a plan and scope committee which became, upon the adoption of the Constitution and By-laws, the Council of the Society. Joseph O'Dwyer, the first vice-president, distinguished for all time by his discovery of intubation, died a short time after the organization of the Society. Of the eleven founders, T. Passmore Berens lived to be well known for his otolaryngological knowledge and skill. He was a surgeon of the Manhattan Eye and Ear Hospital. H. Holbrook Curtis was famous for his treatment of voice affections of singers. He was a devoted member of the Society and served as its librarian for many years. Lewis A. Coffin died three years ago, remembered by us all for his keen wit and ready rejoinder. No one was more to be feared in debate than he. No one took a more active part in the deliberations of the Society. He was distinguished for his contributions to nasal accessory sinus disease, especially in children. Like Curtis, he had been president of this Society and was for a long time a throat surgeon in the Manhattan Eye and Ear Hospital. There followed him, two years ago, his boyhood friend, Wendell C. Phillips. It seems hardly possible that he is gone, he was so much one of us. With Myles, Curtis and Coffin, he was most active in the organization of the Society, succeeding Miles in the office of secretary, and in 1907 became its president. Phillips later became president of the Medical Society of the State of New York and finally received the highest honor that can be bestowed upon a medical man in this country by his election as president of the American Medical Associa-

tion. He had a delightful personality, a commanding appearance, with his snow white hair, rosy cheeks and blue eyes. He was an excellent after-dinner speaker, as was Coffin, and was always in demand. Finally, only last February, the first President of the Society and for life Honorary President, Edward Bradford Dench, passed on after years of suffering. Dr. Dench was a voluminous contributor to the meetings of the Society, having written for its meetings not less than twenty-five papers, many of them of the highest merit. He was generally recognized as the leader of his profession in New York and his loss is felt by us all.

Of charter members still living, Robert C. Myles was for many years one of the outstanding otolaryngologists in New York. He was the first secretary of this Society, and in 1901 became its president. For some time he has lived in retirement, but is still deeply interested in the welfare of the Society. Dr. William L. Culbert continues in active practice in New York City. Dr. Dwight L. Hubbard, who was compelled to retire from medicine on account of ill health, is a resident of Denver and expects to be present at this meeting if his health permits. He also retains a warm interest in all that concerns the Triological.

While others soon joined their ranks, and while it would be a gross omission to fail to mention the invaluable assistance and support given by those from other cities, including Makuen, Kyle, Packard and Smith of Philadelphia, Richardson of Washington, Richards of Fall River, Pierce of Chicago, Goldstein and Loeb of St. Louis, Stucky of Lexington, Levy of Denver and many others, it truthfully can be said that except for the wise planning and judicious guidance by this little group of founders, in the early days, when the breakers were rolling high, the tiny craft would have gone under. Few of us can ever know the courage and devotion of these men, and of the others who shortly after attached themselves. It is to them are due the credit and honor for what the Society is today.

What now was the object or objects of this new association? This is best answered in the language of Article II, of the original Constitution: "The object of this Society shall be the promotion of the science and art of medicine, especially in its relation to laryngology, rhinology and otology, the development of its members in original scientific work and the promotion of social intercourse."

It required only a short time to show that a society having such objects for its existence manifestly was needed. From the founders'

meeting of eleven present and eleven more who signified their intention to join, the Society grew to number 124 at the second annual meeting, 153 at the third meeting, 165 at the fourth, 226 at the seventh and 305 at the twenty-first, and this morning, at the forty-second, just short of 400.

Such a rapid growth is a cause for gratification to us all. Numbers alone, however, mean but little. How far, in the light of the several objects just stated, has the Society justified its existence? At the first it may be said there was a question in the minds of some whether it should or could continue. Some regarded it as antagonistic to the older special societies. This was soon disproved. Each year its ranks have been invaded for recruits for these organizations, until today, with no desire of boasting, it can be truthfully said that the American Laryngological Association and the American Otological Society are composed largely of those most loyal and devoted to the child who this June celebrates its forty-second birthday.

These two societies by the nature of their organization are limited in number. This Society welcomes and seeks all in our specialties who are doing honest work. In the words of Dr. Myles, in his inaugural of 1901, "Our faith in honest work is akin to religion—the doors of our scientific temple are open to all who have the ethical character, attainments and practical experience that are requisite in our high calling."

Of the threefold objects for its existence, the results attained in the first, i. e., "the promotion of the art and science of medicine, especially in laryngology and otology," we can speak in no uncertain terms at the end of forty years. There have been read before the Society at its annual and sections' meetings and printed in our Transactions fully 2,000 papers. Many of them are of the highest value and represent real contributions to medical science. The limitation of time makes it impossible to even mention most of these. A few are so outstanding because of their originality or unusual merit that they must receive all too brief notice. The first of these is a masterly paper by the first secretary of the Society, Robert C. Myles, on the treatment of diseases of the nasal accessory sinuses, read at the first scientific meeting of the Society in 1896, and with two previous papers read before the New York Academy of Medicine in 1893 and 1895, constitutes the earliest serious contribution to nasal accessory sinus disease in this country. The following year H. Holbrook Curtis contributed a classical paper on "The Cure of Singer's Nodules" by nonsurgical methods. The same year Dench made one of the earliest,

if not the earliest, contribution on "Labyrinthine Phenomena Depending Upon Middle Ear Disease, and Their Relief by Local Treatment." He was the first also, if not the first to contribute a paper on "Ligation of the Jugular Vein for Thrombosis of the Lateral Sinus With Report of Three Successful Cases." At the Boston meeting of the Society in 1906, those of us who were present remember the thorough way in which Coakley presented, for the first time in this country, a paper on "Frontal Sinusitis, Diagnosis, Treatment and Result," based on a long series of cases. This was prior to Killian's visit to this country in 1907, and Coakley stated that he had not at that time performed the Killian operation. In 1904 McKernon was the first to report a case of "Primary Thrombosis of the Jugular Bulb." The following year, 1906, Loeb presented an exhaustive study of the anatomy of the nasal accessory sinuses and precursor of his classical two volume work on the subject which appeared some years later. Nothing ever was presented before the Society that received more favorable consideration than the paper by John D. Richards, in 1907, on the Surgery of the Labyrinth. Nothing on the subject that has appeared since that time has exceeded it in value. Our esteemed fellow member Mosher began his active connection with the Society in 1903 in an authoritative paper on "The Tonsil at Birth," presented as his candidate's thesis, to be followed four years later by an anatomic study on the "Premaxillary Wings and Deviations of the Septum," and about the same time Canfield presented a paper on the "Intranasal Route for the Cure of the Disease of the Antrum of Highmore." The following year Ballinger, whose presence at our meeting was always an inspiration, read a paper on "Submucous Resection of the Cartilaginous Septum," with a description of his famous swivel knife. Dench, in 1912, was one of the first to report a successful operation for brain abscess. At the same meeting, 1912, Sluder presented his paper describing his method of tonsillectomy. Two years later, Lynch, as his candidate's thesis, read before the Society a paper on "Suspension Laryngoscopy," which established his position for all time. At the 1917 meeting, Haskin gave a practical demonstration of suction in the treatment of diseases of the nose and throat. This was the first national demonstration of what has since become an indispensable and invaluable agent in special as well as in general surgery. In 1922, Fowler set forth the results of his studies with Mr. Wegel and others, which resulted in the construction of the Western Electric audiometer, in general use today. The lamented Leon White, in 1921, made a valuable contribution on the "Etiology and Pathology of Loss of Vision From the Disease of the Accessory Sinuses." In the Triological Hall of fame

certainly our honored fellow-member, Chevalier Jackson, deserves a place. In addition to the many papers on his chosen specialty which he has read before the Society, he, in 1923, presented an exhaustive paper reporting "1155 cases of foreign bodies in the air and food passages." In 1925, scarcely ten years ago, Mackenty presented his paper on "Laryngectomy for Cancer of the Larynx." In the five years which were allowed him to live after this most valuable contribution, he gave total laryngectomy an assured position in the treatment of this usually fatal disease. Finally, six years ago, Eagleton was one of the first, if not the first in this country, to call attention to the subject of disease of the petrous pyramid," by his paper on "Unlocking of the Petrous Pyramid for Localized Bulbar (Pontine) Meningitis Secondary to Suppuration of the Petrous Apex: Report of Four Cases With Recovery in Three."

Of the second and third objects of the Society, we said in a brief history of the Society at the twenty-first annual meeting in Chicago in 1915, the following: "The second object, that of the development of the members in original work, we have held constantly before us, and striven earnestly to encourage. Numerous prizes have been offered, and the Society has for a number of years set aside a considerable sum of money for a gold medal to reward meritorious original work." The medal was awarded for the first time in 1912 to Samuel J. Kopetzky for his exhaustive work on "Meningitis, Nature, Cause, Diagnoses and Principles of Surgical Relief"; again, in 1915, to Gerhard H. Cox, for his paper on "Experimental Studies of the Effect of Various Atmospheric Conditions Upon the Upper Respiratory Tract," and to Dr. Max A. Goldstein, in 1933, in recognition of his work in the education of the deafened child, while the high standard demanded in the candidates' theses bears witness to the unceasing endeavor of those having the guidance of the Society to fulfill this object. It is in this direction that we should look for still greater advance in the coming years. Every encouragement should be given to develop in still more of our members a true scientific zeal.

Of the last object, "the promotion of social intercourse," we said, in 1915, you who possess the spirit of Phillips and Richardson, among others, who have never in the twenty years missed a single annual meeting, can answer as well as I.

The sweetest, warmest friendships that we know have come out of these annual gatherings. Who can help feeling better for knowing him from Kentucky, who has proclaimed, "No North, no South, no East, no West"? He who has felt the warm hand-grasp of a Duel,

or has been privileged to come to know the honest, simple heart of a George Richards, cannot but be a better man.

We said, "This Society has warm, red blood in its arteries—its age for growth is still before it; death does not stare it in the face—no "mori salutamus" belongs to us for many years. We are all young and few have left us, and yet, even young as we are, we have lost thirty-one members since that 19th day of June, 1895." Since that time, twenty-one years ago, the Society has continued to grow by leaps and bounds. Today it has a membership only a little short of 400. Stucky of Kentucky, Duel and George Richards are no longer with us, but the Society continues to have "warm, red blood in its arteries." Its age for growth is still before it. "Mori-salutamus" has yet no place in its dictionary and never will have if the spirit of the fathers, inherited by the children, continues to be the guiding watchword for the future. With a few exceptions, all the noble company of the founders has passed on. With us who are left to hand our successors the flaming torch belong the problems, the responsibilities and their solution. It is our earnest hope that our ranks will still be open to every man that can meet the demands of membership, which should be so rigid as to prevent any undue further growth in our numbers. Particularly we would urge that the Society resume its practice, as set forth in the second object of its organization, of encouraging the production of original work by the offering of suitable prizes, and finally we earnestly pray that in spite of our enlarged numbers the warm spirit of brotherly love will continue to be cultivated with ever-increasing fervor so that the bonds of fellowship, so noticeable in the early days, may continue to exist without cliques or parties now and in the years to come.

104 East 40th St.

LXV

HERPES ZOSTER OTICUS: REPORT OF THREE CASES*

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WATERVILLE, MAINE

Herpes zoster oticus is a relatively rare condition. According to Hunt's[1] statistics, this diagnosis was made twice in a period of ten years at the Manhattan Eye, Ear and Throat Hospital, six times at the New York Eye and Ear Infirmary, and thirty-three times at the Massachusetts Eye and Ear Infirmary. In Hunt's[2] surveys of the literature he found forty-seven case reports of herpes zoster oticus. During the last ten years this diagnosis was not made at the Manhattan Eye, Ear and Throat Hospital, at least upon house cases. Statistics for the out-patient department are not available. The records of the New York Eye and Ear Infirmary and of the Massachusetts Eye and Ear Infirmary each show six cases during this same period, the latter institution having four as house cases and two as out-patients. Since Hunt's several papers, a number of contributions on this subject have appeared in the literature from time to time, mainly in the form of case reports. The papers of Dabney,[3] Dennis[4] and Sears[5] were especially notable. Fenton's[6] contribution, in 1934, based upon modern studies in immunology, should be referred to for a present-day conception of the fundamental factors involved in this condition.

In 1900 Head and Campbell[7] concluded, from postmortem studies, that the pathologic picture in herpes zoster was one of hemorrhagic inflammation of the posterior root ganglia, together with degeneration of nerve cells and of fibers in the posterior column of the spinal cord. While the exact etiology is still in doubt, it is the concensus of opinion that the causative agent is a filterable virus. Teague and Goodpasture[8] showed experimentally that the virus can pass along the nerve from some inoculated area to the spinal cord and brain, the transmission being along the axis cylinder of either motor, sensory or sympathetic fibers. According to Hunt,[9] the ganglia of the fifth, seventh, eighth, ninth and tenth cranial nerves, homologues of the posterior spinal ganglia, are embryologic outgrowths from the neural ridge and, "in common with other ganglia having this origin, are susceptible to the specific inflammation of herpes zoster." He con-

*Read before American Otological Society, Detroit, May 29, 1936.

sidered herpes zoster oticus, typically, as a primary involvement of the geniculate ganglion, with varying secondary involvement of adjacent ganglia.

Hunt divided herpes zoster oticus, according to clinical manifestations, as follows:

A. Herpes oticus.

B. Herpes oticus with facial palsy.

C. Herpes oticus with facial palsy and hypo-acousis.

D. Herpes oticus with facial palsy and Ménière's syndrome.

The prodromal symptoms may be confined to malaise, headache and slight fever, or may be more severe with nausea, vomiting and increasing pain. Pain is the outstanding symptom and usually precedes the appearance of the herpetic eruption, although Nattkemper[10] reports a case with typical eruption and facial palsy but no pain. The eruption is vesicular and confined to definite areas, depending upon the ganglia involved. This distribution was carefully worked out by Hunt.[1] According to him, the anterior part of the auricle and anterior portion of the canal and tympanum, supplied by the fifth nerve, may be affected through involvement of the Gasserian ganglion. The most frequent location of the eruption, the posterior portion of the canal and tympanum, concha, antihelix, fossa of the antihelix and tip of the antitragus are innervated from the geniculate, the glossopharyngeal and the vagal ganglia. With involvement of the petrous ganglion of the ninth and the jugular ganglion of the tenth, we may also expect to find vesicles homolaterally in the pharynx and larynx. Palatal paralysis may accompany the eruption. Recurrent laryngeal paralysis has been noted in a case report by McKenzie.[11] A case showing both palatal and laryngeal paralysis was reported by Worms and Lieux.[12] Eruption on the posterior portion of the auricle and surface of the mastoid indicates participation by the second and third cervicals. Involvement of the auditory ganglia produces symptoms of deafness, tinnitus and vertigo. While most cases are unilateral, a few showing bilateral involvement have been reported.[6] Stewart[13] had a case with eruption, facial palsy and auditory symptoms on the left, together with involvement of Scarpa's ganglion on the right.

Facial palsy is common in the more severe cases. Sharpe[14] explains the frequency of this by showing that the ganglia of the seventh, as well as the eighth, ninth and tenth nerves, have no fibrous capsule and are in close relation with the nerve fibers, so that slight inflammation of the ganglia tends to involve these adjacent fibers.

Paralysis will be complete in all divisions and involve the sensation of taste through the chorda tympani. Vesicles are often seen on the anterior two-thirds of the tongue. Rebattu[15] and his co-workers report a case of bilateral facial palsy, in which the paralysis on one side was due to herpes zoster, and on the other to a suppurative parotitis. While the facial palsy usually follows the appearance of the eruption and generally accompanies any auditory symptoms, all sorts of variations have been observed. Facial palsy preceding the eruption has been noted by Closier,[16] Haymann[17] and by Lemaitre[18] and his associates. Guthrie[19] reports a case with initial facial palsy and another with auditory symptoms but with the eruption confined to the forehead, cheek and chin, the type referred to by Hunt as herpes facialis with acoustic complications. Tinel, Baruk and Casteran[20] had a case with facial palsy and the vesicles confined to the tongue. Closier[16] and Haymann[21] report cases in which auditory involvement was not accompanied by facial palsy.

Hunt[22] considered the possibility of Ménière's syndrome being herpetic, due to involvement of the auditory ganglia, without eruption. Watson[23] and Key-Aberg[24] both reported cases which showed no eruption but which were considered otherwise typical. The latter considered the condition more properly as a polyneuritis of the internal auditory meatus, either with or without herpes. Hunt[2] also suggested a similarity of this condition to Bell's palsy. However, Aitken and Brain[25] obtained only four positive reactions from the complement fixation test in a series of twenty-two cases of Bell's palsy, while eight cases of definite herpes zoster with facial palsy were all positive. Souques[26] was of the opinion that certain cases of facial paralysis may be due to herpes zoster oticus and go unrecognized because of the scanty or overlooked eruption. Fenton[6] says that pain accompanying facial palsy should always suggest herpes zoster.

It is now considered that the lesion is not a ganglionitis, in any limited sense, but an infectious process involving all nerve structures, ascending or descending, and extending to the cortex in some cases. "It may be likened to lightning—it zigzags from nerve to nerve, and we never know where it will strike"—(Mosher). Recently a specific antibody has been found in the blood.[25] A great deal of evidence has been produced linking this condition with herpes simplex, encephalitis and varicella;[27] [28] although some of this work is apparently contradictory, and the later investigations tend toward a specific distinct virus as the cause. Emerson,[29] Beck,[30] Jaehne[31] and others have stressed the importance of focal infection, especially in the tonsil or sinuses. Exposure to cold, excessive heat, exhaustion and shock

have been mentioned as predisposing factors. Apparently it is a matter of lowered individual resistance to invasion by the virus.

The prognosis is generally favorable, although auditory function may be permanently damaged or lost. Apparently the spiral ganglion is least resistant to the virus and is the slowest to recover. Consequently one can never be as sanguine about return of hearing as of facial and labyrinthine function.

The treatment is symptomatic, directed largely towards efforts for the relief of pain. Any definite focal infection which might have any bearing on a case should be cleared up if possible. Dabney[3] reported temporary alleviation of pain from the use of high frequency electricity. Bertoin,[32] in 1924, suggested the possibility of operation on the geniculate ganglion by a method analogous to that used for resection of the Gasserian ganglion. This would hardly be considered in the light of the present-day conception of the condition. Relief of pain by application of cocain to the region of the sphenopalatine ganglion has been reported by Ruskin,[33] Badeaux[34] and by Fenton.[6] Foreign protein injections and autohemotherapy has been advocated by Rebattu[35] and his co-workers. Friedenwald[36] reports a case of herpes zoster ophthalmicus successfully treated with convalescent serum, obtained from a patient recently recovered from a severe attack of perineal herpes zoster. During the past few years a number of writers[37 38 39 40 41 42] have reported successful results from the use of roentgen-ray therapy. This was used in two of our cases with apparent benefit.

CASE 1.—A woman, aged 27, had intense pain in and about the left ear for five days, together with nausea and vomiting, but no vertigo, deafness, or tinnitus. She was of rather a neurotic type. Examination showed an area of reddish vesicular eruption, quadrilateral in shape, over the mastoid, and several blebs on the posterior canal wall. Membrana tympani normal in appearance. Hearing normal. Nose, throat and larynx normal. Temperature 98.6. Pantocain ointment was prescribed. The pain subsided and the blebs disappeared within a few days.

This case falls within the first and simplest of Hunt's clinical subdivisions. It is similar to Vail's[13] case in that the eruption was confined to the mastoid and the posterior canal wall, and that there was no facial palsy nor auditory disturbance. It is possible that this type is most frequently overlooked or wrongly diagnosed as eczema, erysipelas, frostbite or dermatitis venenata.

CASE 2.—A man, aged 67, had been in poor general health with gradually increasing deafness and tinnitus since an attack of pneumonia five years before. He was brought to the office in an extremely ill condition, complaining of intense pain in the left ear, of seven days' duration, following exposure to severe cold.

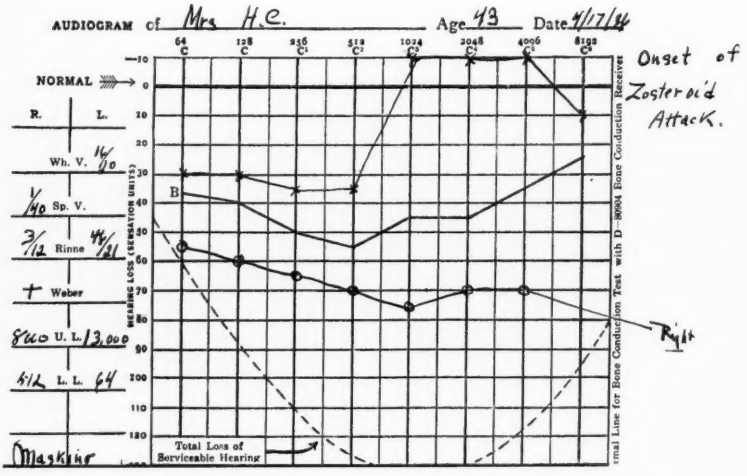
For two days he had had nausea, vomiting and vertigo. Examination of ears, nose, throat and larynx was essentially negative. Teeth were missing. There was no spontaneous nystagmus. Hearing test: Whisper, right 2 feet, left 1 foot. Rinné: right 35/12, left 33/12. Monochord: right 8,000, left 12,000. Low limit: right 64, left 128. Weber lateralized to the left. He was sent to the hospital. When seen two hours later a number of small blebs, containing thin colorless serum, were noted on the left auricle, confined to the concha, antihelix, and fossa of the antihelix. Diagnosis of herpes zoster oticus was made. The next day blebs were found just above the auricle in the temporal region and on the posterior canal wall; and on the following day, on the membrana tympani.

Roentgenologic examination: Mastoids normal, sinuses normal, retained left upper molar root. Ophthalmologic examination (H. F. Hill) negative, except for advanced retinal arteriosclerosis. General physical examination (J. O. Piper) essentially negative except for peripheral arteriosclerosis. Kahn test normal. Urine normal. Blood: Hgb. 90, R. B. C. 4,680,000, W. B. C. 7,700. Neutrophiles 68, small monocytes 32. Cells normal in size, shape and color. Platelets normal.

Three days later the membrana tympani became swollen, with landmarks obliterated, and was incised, with resulting serosanguinous discharge. Culture from this showed a growth of staphylococci and streptococci pyogenes. Temperature which had averaged 101, now became normal and remained subnormal to normal throughout the rest of his stay in the hospital. The pain subsided following incision of the tympanum. The blebs persisted on the auricle and the canal, but disappeared from the temporal region. Four days later the patient had a relapse with marked pain, vertigo and vomiting. There was a definite horizontal nystagmus to the left, and a left facial weakness, which became a complete paralysis in three days. Patient was drowsy and difficult to arouse, lying on his right side. Moving him from this position of election caused vomiting. Hearing test now showed no hearing for voice, tuning forks, or monochord in the left ear. Weber lateralized to the right. Labyrinth tests were not done at this time, or later, owing to the patient's condition. Lumbar puncture showed a clear fluid, normal pressure, fifteen cells, all lymphocytes. Globulin was normal, sugar slightly decreased. A retained dental root was extracted (J. R. Mabee). The vertigo disappeared and the patient improved for ten days, after which there was a recurrence of pain and vertigo, and he became mentally cloudy and delirious. There was an increase in the middle ear discharge. The pars tensa appeared to have practically melted away, leaving the handle of the malleus and the long process of the incus exposed in the middle ear. X-ray treatments were instituted with relief of pain. The encephalitis cleared and there was a slow but gradual improvement with occasional slight remissions. At one time blebs appeared for a few days over the mastoid. Those on the auricle disappeared, leaving an area of necrosis with bare cartilage in the region of the antihelix and its fossa. The auricle became swollen and thickened, having the appearance of a perichondritis. This gradually subsided. The middle ear became dry and the membrana tympani gradually regenerated until there was only a small cicatrix in the superior posterior quadrant. The vertigo and nystagmus disappeared and the facial nerve entirely recovered. The area on the auricle was practically healed when the patient was discharged 73 days after admission. On discharge, hearing tests showed: no hearing for voice, tuning forks, or monochord by air conduction. Weber lateralized to the right. Monochord heard by bone conduction in the left ear at 9,000. Recent hearing tests show no hearing in the left ear. This is apparently permanently damaged.

This case is typical of the last of Hunt's subdivisions, herpes oticus, with facial palsy and Ménière's syndrome, and similar to Emerson's[11] case in that there was an accompanying encephalitis. Arteriosclerotic individuals are apt to be quite susceptible to the virus of herpes zoster. At no time were there any vesicles noted on the tongue, in the throat or the larynx, nor was there any palatal paralysis. Recovery was complete except for hearing. Labyrinth tests would have added to the completeness of the record, but at no time, even on his discharge from the hospital, did it seem advisable to subject him to them. How much the removal of the dental root had to do with his recovery is a matter of conjecture, as is the use of roentgen therapy.

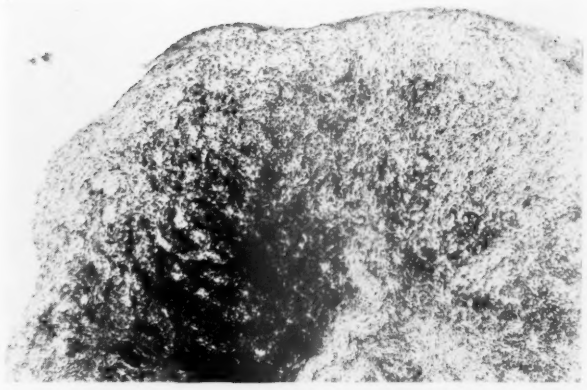
Having already arrived at a diagnosis of herpes zoster oticus I hesitated to incise the membrana tympani, doing so only because of the very evident picture of a middle ear involvement. The rationale of this seemed indicated by the drop in temperature and the temporary relief of pain. Secondary or coincident otitis media has been reported by Gelle,[44] Svenson,[45] Szende,[46] Parthiot,[47] Chavanne[48] (who also quoted reports by Guillermin and Migayron, and by Ramadier), and by Maybaum and Druss[49] in postmortem findings. Sears[5] suggested the possibility of vesicular inflammation of the mucosa of the paranasal sinuses and also of the middle ear in these cases. Guttman,[50] in 1926, stated that most of the otitic complications of influenza were herpetic rather than true otitis media, and warned against incision, but in the cases described by him the tympanic membrane itself showed no pathology unless it was the site of one or more vesicles. It seems probable that influenza is also due to a filterable virus and is quite akin to herpes zoster. Blebs on a red swollen tympanum and on the canal wall near the annulus have long been considered typical of so-called influenzal otitis media. May we not get a secondary bacterial infection in mucosal lined cavities such as the middle ear or the sphenoid sinus, superimposed upon the original virus infection? Autopsy findings on cases during the 1918 epidemic revealed pus in the sphenoid sinuses and cultures from these and from the middle ear repeatedly showed growths of streptococci. Leucopenia is characteristic of influenza, but many cases which are clinically typical soon show a leucocytosis, which would seem to indicate the possibility of secondary bacterial infection. I am bound to agree with Vail, who, in discussing Fenton's[6] paper, said he could see no contraindication to incising the tympanic membrane if it presented definite pathology. The bacteriologic report in this case certainly suggests a secondary otitis media, while the rather unusual picture of the middle ear in its


AUDIOGRAM of Mrs H.C. Age 43 Date 4/17/44
64 C 128 C 256 C1 512 C2 1024 C3 2048 C4 4096 C5 8192 C6
Onset of Zosteroid Attack.
NORMAL
R. L.
Wh. V. 14/40
1/40 Sp. V.
3/12 Rinne 44/21
Weber
8000 U. L. 13,000
4/12 L. L. 64
Maskine
HEARING LOSS (SENSATION UNITS)
B
Right
Total Loss of Serviceable Hearing
mal Line for Bone Conduction Test with D-30004 Bone Conduction Receiver

Case 3. Audiogram at onset of zosteroid attack. (Test in sound-proof room.)

subsequent course keeps the idea of a primary vesicular involvement in mind. There was apparently a necrosis of the drum. The pars tensa seemed to melt away, leaving the handle of the malleus and the long process of the incus exposed in front of a beefy red promontory. As the condition subsided there was a complete regeneration with only a small cicatrix to be seen. The process seemed somewhat analogous to that which so often occurs following vesicular eruption on the auricle.

CASE 3.—A woman, present age 40, of rather a frail neurotic type, first came under my care twenty years ago. She had had a slight chronic suppurative otitis media since childhood. Four days before she had sustained a fracture of the skull as a result of an automobile accident, and had developed a facial paralysis twenty-four hours later. X-rays showed the line of fracture extending from the right parietal region through the superior posterior wall of the right external auditory meatus. The fracture occluded the canal in such a manner as to prevent a view of the middle ear. There were blood, pus and cholesteatoma in the canal. Except for the facial paralysis neurologic examination was negative. The voice was heard at one foot. Rinné was 15/15, and the Weber was lateralized to the affected side. Radical mastoid operation with secondary skin graft was performed. The cavity epidermatized well and the facial paralysis cleared in two months. Six months later she began to have intermittent attacks of slight vertigo. The condition remained the same for eleven years (nine years after the accident) when she developed periodic attacks of intense burning pain, together with blebs containing thin bloody fluid, on the concha and in the scar of the Korner plastic. A diagnosis of herpes zoster oticus was then made. These attacks have continued ever since, com-



Case 3. Photomicrograph of biopsy specimen from radical cavity. Fibrous polyp.

ing on at varying periods. Nine years ago she had an attack of thoracic herpes zoster.»

Tonsils missing, X-rays of sinuses and teeth normal. Fundi and fields reported normal. Basal metabolism test, 7. Kahn test normal Hearing test (right): conversation, 7 feet. Rinné 15/15, monochord 12,000, low limit 256, Weber lateralized to the right. Rhomberg negative. Vertigo on head being bent backward. Walking with eyes closed gives sensation of going to the left. No spontaneous nystagmus. Pointing reactions normal. Normal response but rather excessive vertigo to both rotation and caloric tests. Fistula test has always been positive.

Eight years ago a narrow furrow was noted just posterior to the facial ridge. This had been previously well epidermatized. This furrow gradually increased and began to show a soft projecting growth, with a leakage of colorless fluid. Touching this area caused vertigo. Pathologic report, following biopsy from this growth, was as follows: "Dense fibrosis, lymphocytes and plasma cells. Fibrous polyp with chronic inflammation." Two years ago I removed this polypoid growth from the radical cavity. This was very fibrous, coming away in three lobes, each about the size of a pea. No bare bone was felt underneath. The area healed except for a distinct fissure, remaining so for about eighteen months, when the polyp began to recur. Pressure on this still causes vertigo. For the past few years the attacks of herpes have been less frequent, being more evident in cold weather and usually associated with the menstrual period. For the most part the pain has been controlled by a mild cocain ointment, but when especially severe X-ray treatments have been given with relief lasting several months.

In this case the skull fracture invaded the aqueductus fallopii, causing a facial paralysis which was later relieved by operation. The fracture through the petrous pyramid may never have entirely healed, although recent x-rays fail to reveal any evidence of it. After several years the epidermatized area over this fracture gave way allowing escape of perilymph, and the gradual formation and projec-

tion upward of a fibrous polyp from the bone edges. According to Vail,[51] cases of herpes zoster following injury are done to the entrance of specific infection through a septic wound. The fracture might have provided a portal of entry, but it is difficult to assume that such a chronic course, with periodic remissions dating back eleven years, could be due to a specific infectious disease. The occurrence of a thoracic herpes zoster at one time is of interest. Probably there is some inherent or constitutional factor predisposing to this condition. Dench,[52] in his contribution to Burnett's textbook in 1896, noted that herpes zoster oticus was most likely to attack individuals of a neurotic temperament. Neither the facial palsy nor the labyrinthine irritation can be attributed to the herpes. They both antedate the appearance of pain and the eruption by nine years, and the former recovered after operation. The hearing has been quite satisfactory for a case having had a radical mastoid operation, except during the herpetic attacks, when there is a temporary diminution. Up to the present this has returned to its former level after subsidence of the attack. Forty-one years ago Grindon[53] differentiated secondary zoster from the true idiopathic type. He termed the former, caused by some local pathologic state, "zosteroid." Among the causes mentioned by Grindon were trauma, pressure on nerve trunks, chronic peripheral irritation, infiltration of nerve or ganglion from a neoplasm, or some irritating substance, such as lactic or uric acid in the blood. He considered that the recurring forms were of this type. Veyssi and Nicolas[54] report a case with repeated attacks of vesicular eruption, facial palsy and auditory symptoms, and referred to another case of similar type except for the facial palsy. They felt that the condition was due to lesions in the fallopian canal following an old suppurative otitis media. They regarded this as a zosteriform eruption rather than a true herpes zoster. Chronic irritation incident to the labyrinthine fistula might seem to account for the recurring herpetic symptoms in my case. It is unfortunate that we have been unable to obtain material for a complement fixation test, as this might have been of added interest.

The neurologic manifestations incident to a herpes zoster are the result and not the cause of this condition. The clinical picture may be produced by a varying etiology. It may be a filterable virus, possibly reinforced by secondary bacterial infection, or it may be trauma, chronic irritation, or pressure on nerve trunk or ganglion. If we are to regard herpes zoster as a true specific infection, as indicated by scientific investigation, the latter etiologic type must be considered as zosteroid, reviving Grindon's differentiation.

CONCLUSIONS

1. Herpes zoster oticus is a rare condition. Some cases, especially of the simplest type, may be overlooked or wrongly diagnosed.

2. Hearing function is that most apt to be permanently damaged.

3. There may be middle ear involvement, due to secondary infection, which will require, at least, incision. The usual course of otitis media may be altered by the manifestations of the herpes.

4. Roentgen-ray therapy may be beneficial, especially in alleviation of pain.

5. The same clinical picture may be produced by a variety of causes. Application of recent immunologic studies may better differentiate true herpes zoster from the zosteroid type.

PROFESSIONAL BUILDING.

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LXVI

A SUGGESTED OPERATIVE PROCEDURE FOR THE RELIEF OF STENOSIS IN DOUBLE ABDUCTOR PARALYSIS: AN ANATOMIC STUDY*

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NEW YORK

It is with a great sense of temerity, after a long series of failures by others, that this operative procedure, based on an anatomic study, is suggested for the relief of the laryngeal stenosis caused by double abductor paralysis of the vocal cords.

It has been interesting and instructive, in going over the history of the various operative procedures, to note the line of reasoning which prompted these various operations and the end result obtained. The mere multiplicity of operations for this condition is indicative of the poor end results achieved.

Naturally the primary object has been to increase the airway, thus overcoming the stenosis. Some have attempted the reinnervation of the larynx by simple suturing of the severed ends of the recurrent laryngeal nerves,[1] or by some form of anastomosis of this nerve with the vagus (from which it comes), facial,[2] descendens noni,[3] or the phrenic nerves.[4 5 6]

Others have attempted to increase the airway by removing the obstructing agents (vocal cords), or by trying to displace these cords laterally.[7 8 9 10 11 12]

Recently Hoover[13] proposed a submucous resection of the vocal cords. This operative procedure probably has been the most promising in obtaining the desired results. Strange to state, I had worked out this operation on about thirty larynges, when Hoover reported his work along the same lines. Technically it is not as simple as one would think, after first reading his article.

In this anatomic study, full consideration has been given to the various forces acting to keep the larynx as it normally is. The sphincters[14] of the larynx are the thyro-arytenoideus, crico-arytenoi-

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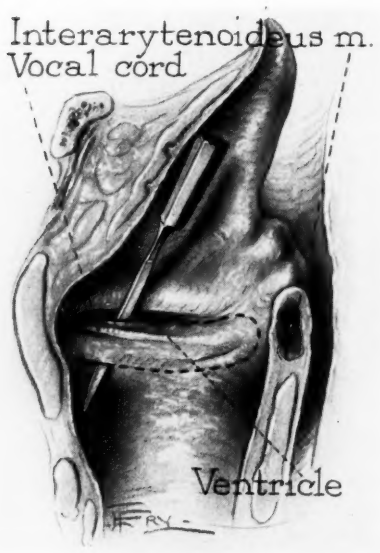

Fig. 1. Start of incision. Knife is plunged through floor of ventricle, coming out subglottically.

deus lateralis and the inter-arytenoideus muscles, while the dilators are the crico-arytenoideus posticus muscles. These groups of muscles are supplied by the recurrent laryngeal nerves. According to Semon's law, a destructive lesion of the nerve, whether along its course or in the medulla, first causes a paralysis of the abductor muscles, and later, of the adductors. Hence it is the unopposed actions of the thyro-arytenoideus, crico-arytenoideus lateralis and inter-arytenoideus muscles, in these paralyses, which draw the vocal cords to the midline position and produce the stenosis. Later on, when all the muscles are paralyzed, the cords may assume the cadaveric position, partially relieving the stenosis.

In operative procedures on the vocal cords, whether they be stripping, complete removal or thyrotomy, there is a replacement of the tissue removed, so much so that in time a new or so-called adventitious cord is formed, occupying the position of the original one. What is the modus operandi which makes this possible?

It must be assumed that the vocal cord has two points of attachment: one is the anterior commissure and the other is the vocal

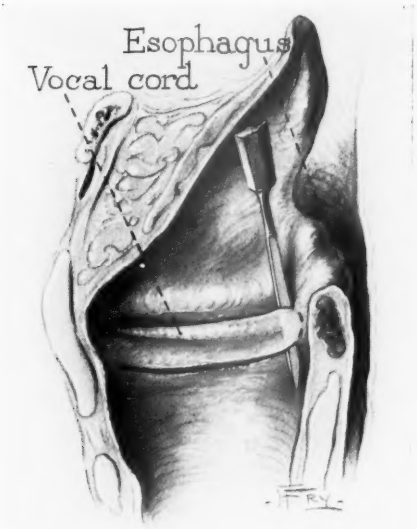

Fig. 2 End of incision. Knife has been carried internal to vocal process and body of arytenoid cartilage.

process of the arytenoid. It is the constant pull between these points which limits the cords to the midline.

In operated cases, in which tissue has been removed (thyrotomy, for example), there is considerable granulation tissue formed to fill in the defect caused by the operation. While this filling-in process is going on there is a growth of mucous membrane from the denuded edges produced by the operative procedure. This growth of mucous membrane is towards the center of the deficient area, now being filled in with granulation tissue, and in sufficient time this growth of mucous membrane is enough to completely cover this new granulation tissue. Before this happens, however, there is invariably an overgrowth of granulation tissue, enough, in fact, to protrude well beyond the midline. But as soon as this granulation tissue is covered by mucous membrane there is a recession from beyond the midline until a fixed line is reached, this line being determined by the anterior attachment of the cord and the posterior fixed point (the arytenoid). In other words, the constant pull between these two points limits the new tissue to a line between them. If the arytenoid has been removed, then the second point will be in the region or area where it was, or the new attachment of the posterior end of the cord.

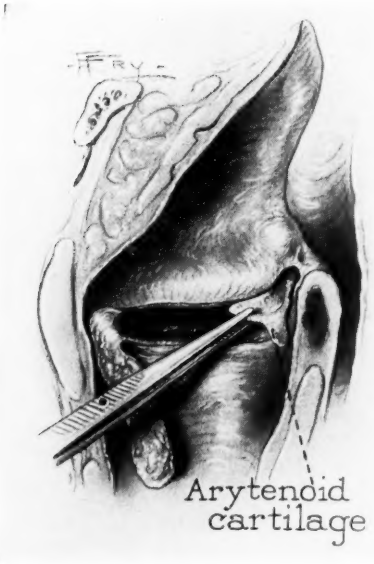

Fig. 3. Removal of the arytenoid cartilage.

If it is true, then, that the position of the cord is determined or influenced by these points, then the shifting of one of these points would change the position of the cord. Predicated on this assumption and Semon's law, the following operative procedure is suggested.

It must be borne in mind that this is an anatomic study, based partly on known facts and partly on theoretical considerations.

It goes without saying that this operation should not be attempted until the permanency of the paralysis has been established. All cases, excepting those resulting from operations or injuries, should have a therapeutic luetic test, even though the blood and spinal fluid be negative for syphilis.

Technic.—In performing the operation a preliminary tracheotomy is indicated. The anesthesia may be either general or local.

The larynx is exposed by means of a laryngo-fissure or thyrotomy. The incision used is the author's[15] modification of the classical one, whereby a V-shaped incision is made through the thyrohyoid membrane after the midline incision has been made through the thyroid cartilage. By means of this incision wide retraction is possi-

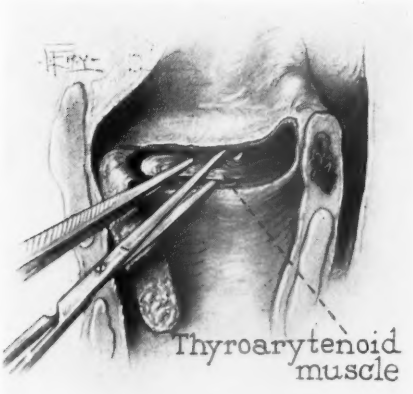

Fig. 4. Removing tissue subjacent to denuded area. Part of thyro-arytenoid and crico-arytenoid (lateral) muscles are included in this removal.

ble, thus obtaining an excellent view of the interior of the larynx and permitting intralaryngeal surgery.

Before proceeding to the operation the trachea is gently packed, thus preventing blood and secretions from reaching the lungs. Then the laryngeal operative field is painted with 50 per cent alcohol.

A small bladed knife is plunged through the floor of the ventricle, close to the anterior commissure, about one-fourth to three-eighths of an inch from the edge of the cord, and coming out about one-fourth to three-eighths of an inch subglottically. With the knife in this position it is carried posteriorly towards the arytenoid, cutting parallel to the vocal cord. When the vocal process is reached the knife is carried internal to it by cutting through the attachment of the vocal cord from it. The knife is then carried parallel and internal to the vocal process and body of the arytenoid until the posterior commissure is reached, at which point the mucous membrane is severed. The arytenoid is then dissected out and removed. Care must be exercised not to leave any part of the arytenoid. We now have a vocal cord to which is attached part of the thyro-arytenoideus muscle detached from its subjacent tissue except at the anterior commissure, where it is still attached. This cord is now gently retracted forward, exposing a denuded area. Through this denuded area the tissues subjacent to the cord are removed down to but not including the perichondrium. In this tissue removed are included the remainder of the internal portion of the thyro-arytenoideus muscle and part of

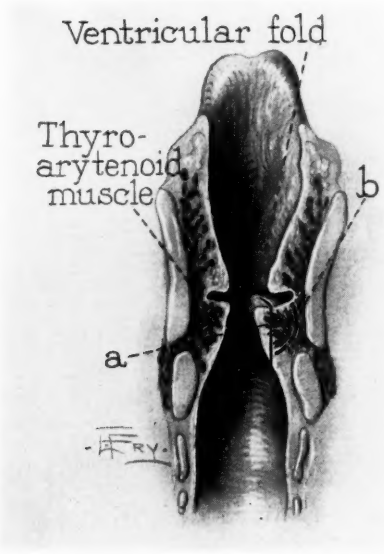

Fig. 5. *(A)* Initial incision. *(B)* Depth to which tissue is removed.

the crico-arytenoideus lateralis muscle. The upper part of the conus elasticus is now undermined for about three-eighths of an inch to permit suturing of the cord. All bleeding having been controlled, the vocal cord is now returned to its original position except posteriorly, where the free end will be more external because of the absence of the arytenoid. It is sutured in this position, using absorbable material and everting the edges.

The interior of the larynx is packed with iodoform gauze, bringing one end out through the crico-thyroid membrane. The thyrotomy wound is closed in the usual way. A feeding tube may be inserted at the time of operation or it may be deferred until later, in order to see whether it is really necessary or not.

The object of this operation is to change the posterior attachment of the cord to a more external one, and at the same time to remove the pull of the thyro-arytenoideus (internal portion) and crico-arytenoideus (lateral) muscles. Naturally the question arises whether one or both cords should be operated upon. If it is felt that the transposition of one cord does not give sufficient space, then both cords should be operated upon. It must be borne in mind, however, that as the airway increases the voice will become worse.

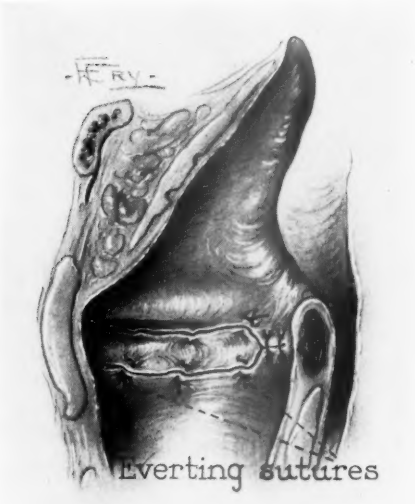

Fig. 6. Suturing of vocal cord over denuded area.

Fig. 7. *(A)* Unoperated vocal cord. *(B)* Operated vocal cord.

What the end results will be I do not know. I feel however, that the procedure merits a trial.

Technically on the cadaver it has been easy. Perhaps performing it on dogs might improve the method and management of such cases. Dr. Kernan has been kind enough to put some dogs at my disposal, and I shall avail myself of the opportunity.

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LXVII

NASAL MUCIN*

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Mucin, an important constituent of many body secretions and tissues, is a glucoprotein containing a complex carbohydrate radical or polysaccharide combined with a protein. This complex polysaccharide, mucoitin sulphuric acid, is made up of four constituents, glucosamine, glucuronic acid, sulphuric acid and acetic acid.[1] Mucins are not identical, as their elementary analyses show, their solubilities are not the same and the degree of viscosity of their aqueous solutions differ. It is thought that these differences probably lie in the protein portion of the molecule, rather than in the carbohydrate complex.

A review of the literature shows that a Russian clinician, Eichwald, 1866, was the first to note that a mucoprotein upon hydrolysis contained a substance that reduced Fehling's solution. Landwehr,[2] 1882, was the first to isolate a polysaccharide from mucin. Later Schmiedeberg[3] secured a different polysaccharide from the mucus of the snail. Neither of the investigators found sulphuric acid or glucuronic acid in the carbohydrate complex. The true nature of the nonprotein radical was identified by the extensive work of P. A. Levene and his co-workers.[1]

In the work reported here mucin was obtained from the nasal discharge of patients suffering from allergic rhinitis. Some of the specimens were prepared by the method of Fogelson[4] and others, by a method essentially that of Chittenden and Gies.[5] There was secured a white amorphous powder which gave the characteristic color reactions of mucin, such as Biuret, Millon's, Hopkins-Cole, Xanthoproteic, Molisch, Liebermann's and the lead acetate test. It did not reduce Fehling's or Benedict's solution without previous hydrolysis in 3 per cent hydrochloric acid. Its reducing power, determined on four different samples, was found to correspond to 24.5 to 26.6 per cent glucose. The reducing power was estimated after acid hydrolysis according to the method of Hagedorn-Jensen. The amount of re-

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ducing substance present in nasal mucin lies within the values for mucins from other sources. The reducing power of mucoproteins from different sources, obtained by different workers, is presented in Table 1.

TABLE 1

THE PERCENTAGE REDUCTION OF MUCIN AFTER ACID HYDROLYSIS

Mucin	*Author*	*Reduction (Glucosamine)* %
Salivary	Muller	32.8—36.9
Submaxillary	Seeman	24.0
Submaxillary, oxen	Muller	20.8—23.5
Gastric juice, dog	Webster and Komarov	12.0—15.0
Gastric surf. epith. dog	Webster and Komarov	31.0—35.0
Ovomucin	Matthews	11.0
Ovomucoid	Seeman	34.9
Ovarian cyst	Zangerle	30.0
Urine	Fleury and Dufau	14.7
Urine, horse	Sahlstedt	30.0
Synovial fluid, beef	Achard and Piettre	8.0
Myxomucin	Ostwald	12.0

Nasal mucin with its high glucosamine content is in all probability a poor antigen. Goodner,[6] in his study on the antigenic properties of glucoproteins, found that glucoproteins do not show strong antigenic properties, and that the greater the amount of extra-proteinous matter in the molecule, the less potent is the substance antigenically. He found that ovomucin which yields 11 per cent glucosamine is a much better antigen than submaxillary mucin or ovomucoid, which yields 24 per cent and 35 per cent glucosamine, respectively.

Analysis gave the following elementary composition:

	I	II
C.	46.81	46.01
H.	7.12	6.69
N.	10.36	10.02
S.	0.64	0.94
Ash	0.45	

Sample I was prepared by the method of Fogelson, II by a method essentially that of Chittenden and Gies. These analyses show that the protein of the nasal discharge is a mucin, but is not identical with any of the mucins found in the literature. The carbon and nitrogen content are appreciably lower than in the other mucins. The difference in the sulphur content of the two samples might be

explained by the method of preparation. In every extraction, not all of the protein would go into solution, regardless of the quantity of the solvent added. This protein, however, took the mucicarmine stain and is therefore a mucin or mucoid substance of different solubility than the one obtained. It is indicated that there is more than one mucin in the nasal discharge. A study of Tables I and II shows that mucins are numerous and vary from tissue to tissue and specie to specie. A complete study of the proteins of the nasal discharge might throw some light upon the protective mechanism of mucus to the organism.

Phosphorus was present in minute quantities, varying between 0.02 per cent and 0.05 per cent. These values are far too low for this element to be an integral part of the mucin molecule. The moist ashing procedure (H_2SO_4+HNO_3) was used and the colorimetric method of Tisdall was performed.

An attempt was made to prepare and identify the constituents of the prosthetic group of mucin. The mucous membrane was stripped from the nasal cavity and accessory sinuses of the calf head, ground in a meat chopper and treated according to the method of P. A. Levene and López-Suárez.[1] The resulting crude salt was very hygroscopic, very soluble in water and required a great excess of glacial acetic acid for precipitation. A white powder was thus obtained. This substance did not give the Biuret test nor did it reduce Fehling's or Benedict's solutions previously to hydrolysis. After heating on a water bath for two to three hours in 3 per cent hydrochloric acid, Benedict's test was strongly positive. The substance prepared is free from proteins and contains a reducing substance. Further identification of mucoitin sulphuric acid was not attempted, as the quantity of the polysaccharide secured was very minute.

TABLE II

THE ELEMENTARY COMPOSITION OF VARIOUS MUCINS

	Hammarsten Beef Submaxillary	Loebsch Beef Tendon	Landwehr Beef Submaxillary	Muller Human Salivary	Webster and Komarov Dog Gastric Juice	Ostwald Myxo-mucin	Achard and Piettre Beef Synovial Fluid
C.	48.8	48.30	49.93	48.86	52.9	50.82	48.6
H	6.8	6.44	7.27	6.91	7.01	7.27	7.0
N	13.3	11.75	13.98	10.70	13.8	12.24	15.4
S	0.84	0.81	0.80	1.38	1.29	1.19	1.62

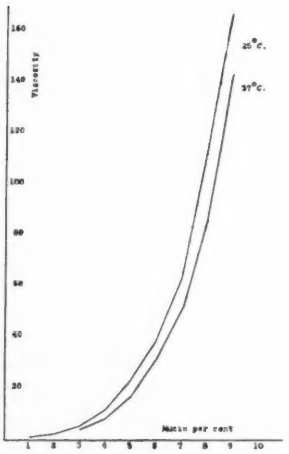

Fig. 1

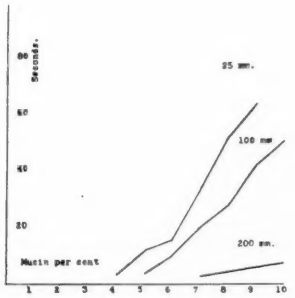

Fig. 2

THE VISCOSITY OF MUCIN.—The viscosity was measured by determining the ratio between the time rate of flow of the mucin solution from a 1 cc. Ostwald pipette and the time rate of flow of distilled water from the same pipette. One per cent to 10 per cent solutions of gastric mucin were made up with distilled water. Measurements of viscosity were made at 25° C. and 37° C. The curve, Fig. 1, rises slowly through the first four solutions; then the viscosity rapidly increases progressively for each increase in percentage. The curve at 37° C. runs parallel to the curve at 25° C., showing a decreased viscosity throughout its course. Therefore an increase in temperature is accompanied by a decrease in viscosity. To determine the influence of ions upon viscosity of mucin, 1 to 10 per cent solutions of mucin were made up with a salt solution having the ionic composition of the blood. The viscosity of these solutions was less than that of the corresponding water dilutions at the same temperature. This is in accord with the work of Deganello,[7] who found that sodium bicarbonate and sodium chloride decreases the viscosity of bronchial secretion and that expectorants had little or no effect. The changes in the physical state of mucin brought about by increased alkalinity and temperature lead one to ponder over the possible connection between these changes and the decreased antibacterial action of the nasal mucous membrane. It is known that allergic nasal secretions and the nasal discharge in the early stage of an acute cold are in many instances very alkaline and have an increased mineral content.[8,9] These factors may play a part in increasing the susceptibility of the individual to secondary infection.

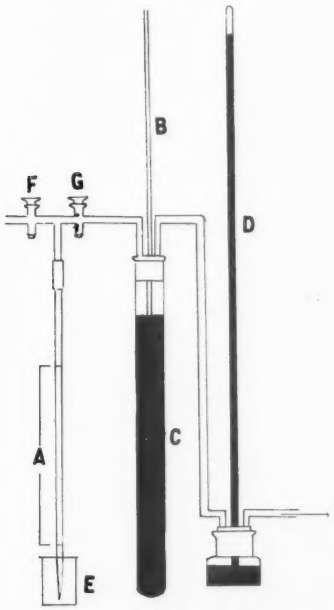

Fig. 3

With stopcock *F* and *G* closed, the amount of vacuum is obtained by adjusting tube *B* in the mercury column *C*. The vacuum is read, mm. of mercury on the barometer *D*. Mucus is placed in the receptacle *E*, stopcock *G* is opened and the time required for mucus to flow distance *A* is noted. The time rate of flow is used to indicate relative viscosity.

Mucin secreted from the nose has a much greater viscosity than the corresponding solution of gastric mucin. A nasal secretion containing 1.26 per cent protein has a viscosity equivalent to an 8.4 per cent solution of gastric mucin. Undoubtedly the precipitation and subsequent drying with ether and alcohol has changed the nature of the substance. Only the thinnest of nasal secretions will flow from a pipette, consequently a constant suction apparatus, Fig. 3, was employed.

As the amount of suction was increased, the more closely the curve approximated a straight line (Fig. 2). At 25 mm. of mercury suction the curve resembles that of a curve secured by gravity, but at a suction equivalent to 200 mm. of Hg., the curve is practically a straight line. Table 3 shows that mechanical stirring of the secretion decreases its viscosity. The viscosity of a thin, clear nasal discharge with low protein content was measured by the suction appa-

ratus. The first attempt was not completed, as the material rose only 12 cm. and stopped. The secretion was stirred and the viscosity again determined. This procedure was repeated several times. Each successive manipulation of the mucus resulted in a decreased time rate of flow. It is suggested that stirring the secretion disturbs the relationship between its water and protein content. Nasal mucus is not homogeneous and only a relative viscosity can be obtained.

TABLE III

THE RELATION OF MECHANICAL DISTURBANCE TO VISCOSITY

Nasal	*Suction*	*Distance*	*Time*
Secretion	200 mm. of Hg.		
Undisturbed	"	None	
First stirring	"	Stopped at 12 cm.	
Second stirring	"	14 cm.	12.0 sec.
Third stirring	"	14 cm.	4.2 "
Fourth stirring	"	14 cm.	3.2 "

SUMMARY

1. Nasal mucin has been prepared and the elementary analysis given. Nasal mucin is not identical with any mucin from other tissues or secretions.

2. The polysaccharide of mucin has been obtained free from protein and identified only by its general physical characteristics.

3. The viscosity of mucin is decreased by increase in temperature and the presence of ionizable salts. The stirring of mucus also decreases its viscosity.

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LXVIII

AURAL MANIFESTATIONS OF LIPOID GRANULOMATOSIS (HAND-SCHÜLLER CHRISTIAN'S DISEASE)*

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In the diseases which have been attributed to disturbances in lipoid metabolism, particularly those associated with the names of Niemann-Pick, Gaucher and Hand-Schüller-Christian, the ear not infrequently shows clinical and pathologic manifestations. This is particularly true with regard to Schüller-Christian's disease. The first two of these diseases have received very little attention by the otologist. Brunner,[1] in 1928, reported a case of Gaucher's disease in which he found the typical cells in the temporal bone; and the writer,[2] in 1932, described the pathologic changes in the ear in Niemann-Pick's disease. The aural manifestations of Hand-Schüller-Christian's disease, however, have been considered along with other symptoms for many years by various authors[3] who dealt with the subject, but it is only in recent years that this aspect has received major consideration in the otologic literature. The recent article by Lederer, Poncher and Fabricant[4] was the first to appear in an American otologic journal. The frequency with which the ear is involved in this condition, and the presence of unusual pathologic features of the case to be presented prompted this report.

For a detailed account of Hand-Schüller-Christian's disease the reader is referred to the excellent works of Pick,[5] Rowland,[6] Chester[7] and Henschen.[8] In each of these entities there is a disturbance in metabolism of different lipoids. Thus in Gaucher's disease there is a storage of cerebroside called kerasin; in Niemann-Pick's disease, an abnormal accumulation of phosphatide with an increase of other lipoids; in Hand-Schüller-Christian's disease, cholesterol and its esters.[8a]

Hand-Schüller-Christian's disease is usually characterized by the following triad, namely, defects in the membranous bones, diabetes insipidus and exophthalmus. The presence of all these symptoms, however, is not essential for the establishment of a diagnosis. While

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the flat bones, particularly those of the skull, are most frequently involved, at times the long bones, tendons and other parts of the body show changes. In the skull the parietal and temporal bones are most frequently affected. The etiology is not known, but it has been ascribed to infections, trauma and endocrine disturbances. The disease usually begins in childhood and frequently runs a long chronic course. Recovery is spontaneous in some instances. Of those which succumb, intercurrent pulmonary involvement is a frequent cause of death.

The pathologic changes vary with the age of the lesion. In the early stages of the disease the so-called xanthomatous lesions are frequently observed. These are conglomerate masses of large pale round and oval cells with fine membranes and pale cytoplasm forming an areolar network containing fat droplets. A large, usually central, nucleus and occasional nucleolus are present. They often clump together and closely resemble giant cells. With the polariscope, doubly refractile bodies, comprised mainly of cholesterol and its esters, are noted within the cells. Because of this fatty content they are known as foam cells (xanthoma cells). They are accompanied by leucocytes, lymphocytes, plasma cells, young fibroblasts (spindle cells) and, together with dilated capillaries, give the picture of granulation tissue; hence the term lipoid granuloma. The granulomata appear in the form of nodules or as diffuse lesions. In the more advanced lesion the foam cells diminish in number and are usually sparsely distributed; the tissue is vascular and undergoes fibrosis and scarring. The origin of the xanthoma cells is not definitely known. The present conception is that they are reticulo-endothelial cells which react to a disturbance in lipoid metabolism, by proliferation and phagocytosis of lipoid material.

CLINICAL MANIFESTATIONS OF THE EAR

Since the parietal and temporal bones are so frequently involved it is natural to expect aural manifestations in a large percentage of cases. Thus, Greifenstein,[9] in 1932, found evidence of ear involvement clinically or at autopsy in 50 per cent of the cases reviewed. A personal study of the cases presented in the literature, with particular attention to aural manifestations, showed that they were referable to practically every part of the temporal bone, namely, the external ear, the middle ear, the mastoid process, the labyrinth, the petrous pyramid, the sigmoid sinus and the dura.

An attempt is made to classify these signs and symptoms according to location in the temporal bone involved.

1. The external ear
   (a) Part of a specific eczematoid skin lesion.
   (b) Ulcerations of skin or bony erosions secondary to disease of mastoid or squama (Fraser's case).[10]
   (c) Concentric atresia of external canal. (Schüller's case 1.[11] Schotte's case.[12]

2. Middle ear

(a) Acute middle ear suppuration is a frequent occurrence (Hand,[13] Rowland case 2,[6] Fraser[10] and others). As a rule, there are no characteristic signs or symptoms. Of interest, however, is the presence of a foul smelling discharge from the middle ear in the case reported by Lichty[14] and Wheeler.[15]

(b) Chronic middle ear suppuration was observed in a number of instances (Schüller,[11] Greifenstein,[9] Kay quoted by Hand[13]). It is often associated with a gritty or cheesy discharge and with polyps or granulations. The hearing impairment is of the conductive type and does not differ from the usual chronic middle ear suppurations (Schüller,[16] Christian[17] and Hofer[18]).

3. Mastoid process. Postauricular swelling secondary to Hand-Schüller-Christian's disease may simulate mastoiditis, particularly when it is associated with middle ear suppuration. This was strikingly demonstrated by Lichty's[14] case, a brief report of which follows. A twenty-one months old child, in the course of illness, developed a tender swelling over the mastoid which was associated with a foul smelling purulent discharge. A diagnosis of acute mastoiditis was made and a mastoidectomy was performed. No pus was found. Instead a yellowish granulation tissue (xanthomic tissue) was encountered in the bone. Lichty was of the opinion that a preoperative x-ray would have ruled out a mastoiditis.

4. Labyrinth. Labyrinthine signs and symptoms are not infrequently present. A total loss of function of the cochlear and vestibular nerves was recorded by Schotte[12] and Greifenstein;[9] a nerve type of hearing impairment by Hochstetter[19] and others.

5. Petrous pyramid. The petrous pyramid was involved in the case reported by Schultz, Wermbter and Puhl.[20]

6. The dura and venous sinuses occasionally show granulomatous infiltrations. They may be revealed at operation by x-ray examination or postmortem. Instances illustrating these findings were reported by Dietrich,[21] Rowland,[6] Henschen,[8] case 2, and Fraser.[10] These granulomatous infiltrations may be primary within the dura or secondary to involvement of the adjacent bone.

Facial paralysis was observed by Fraser. It subsided in his case after a radical mastoidectomy.

Roentgen examination is of considerable value in the diagnosis and localization of the lesions and in the determination of the progress during the course of the illness. The so-called "geographic lesions" in the bone, revealed by x-ray and described by Schüller were often diagnostic. These are sharply defined areas of decalcified bone which assume various shapes and resemble geographic figures. X-ray may assist in disclosing disturbed pneumatization (Greifenstein's disease),[9] clouding of cells due to granulations, destruction of the cells (Rowland), and healing processes in the bone. Thus Greifenstein noted that the granulomatous deposits had a tendency to disappear and to be replaced by very dense newly formed bone. In Wheeler's case an increased density of the petrous pyramid was revealed.

It must be borne in mind that the aural signs and symptoms when present are not always manifestations of Hand-Schüller-Christian's disease *per se*, but may be incidental findings in the course of the illness. This contention is supported by the fact that the disease is most prevalent in children in whom, as is well known, the incidence of upper respiratory infections and middle ear suppurations is relatively high.

REPORT OF A CASE

CASE 1.—An Italian male, aged 48, was first admitted to the medical service of Dr. B. S. Oppenheimer on June 14, 1933, with the following history: Nine years ago, while apparently enjoying good health, he began to consume and void large quantities of water (five to six gallons daily). He received injections of pituitrin for a period of two months, one injection affording relief for the severe thirst for about twelve hours. For the past seven years he had had treatment for the diabetes insipidus, but in spite of this the symptoms have continued up to the present time.

He has had bilateral discharging ears on and off for the past six or seven years, during which time the hearing has been considerably impaired.

Within the past two years he has experienced marked weakness, loss of weight and gradual loss of libido and potency. For the past two months he has suffered almost constant temporal and frontal headaches.

Physical examination: The patient was very pale and appeared to be in a weakened condition. The calvarium was negative. The teeth were in a poor condition, and the gums showed evidences of pyorrhea.

Ears.—There was a scarring of both tympanic membranes. This was noted in the routine examination made by the medical interne. Unfortunately examination (including tuning fork tests) was not performed by the otologic service.

Course in Hospital.—Roentgen examination of the skull was essentially negative except for the finding of calcareous deposits in the falx cerebri. The blood

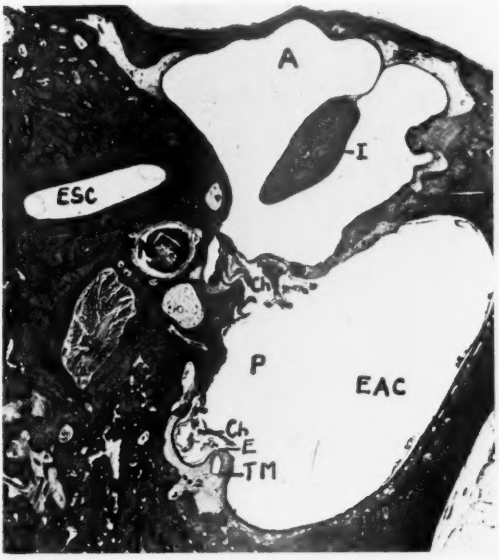

Fig. 1. Vertical section through the aditus. The epidermis of the external auditory canal has grown around the margins of the perforation and has attached itself to the inner tympanic wall with the formation of cholesteatoma. *A*, represents Aditus. *Ch*, Cholesteatoma. *E*, Epidermis. *E. A. C.*, External Auditory Canal. *E. S. C.*, External Semicircular Canal. *F. N.*, Facial Nerve. *P*, Perforation. *I*, Incus. *St.*, Stapedius Muscle. *T. M.*, Tympanic Membrane.

picture was that of a marked secondary anemia with a leucocytosis. Examination for blood cholesterol revealed 144 mgm. per 100 cc. It was felt that this case was one of pituitary neoplasm, possibly Simmond's disease. The patient was discharged six days after admission slightly improved.

He was readmitted November 10, 1933, about five months after his discharge from the hospital with an interval history of progressive loss of weight and weakness, severe vertical and bitemporal headaches, impaired vision and pain in the left arm and right side of the chest.

Course in Hospital.—Fundus examination revealed bilateral hypertensive retinitis.

Xanthomatous infiltrations were noted on both lower eyelids. Tenderness was elicited over both sterno-cleido-mastoids, over various bones of the body, particularly the left lower forearm, where a slight swelling was present. X-ray examination of the bones revealed an increased density of the fifth rib. The findings were interpreted as possibly metastatic lesions in the bones.

An elevation of the cholesterol to 235 mgm. per 100 cc. of blood was found.

The patient gradually went downhill; he had generalized convulsions, became stuporous and ceased twelve days after admission.

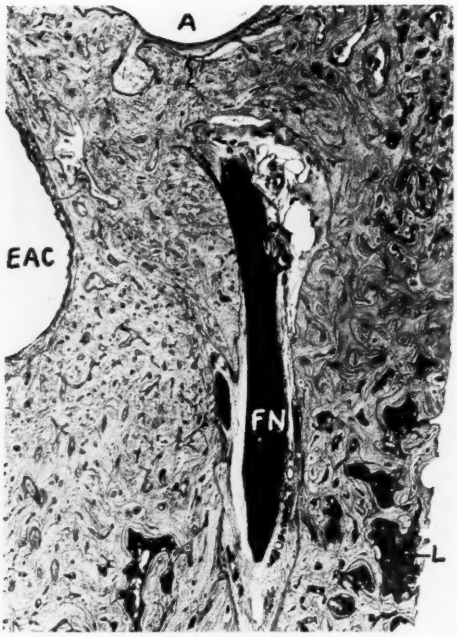

Fig. 2. Section through the external auditory canal and antrum, showing lipoid granulation tissue in the marrow spaces near the facial nerve. The remainder of the bone is sclerotic. *A*, represents Antrum. *E, A. C.*, External Auditory Canal. *F. N.*, Facial Nerve. *L.*, Lipogranulomatous Tissue. (Hemalum and Sudan Stain.)

Necropsy findings. There was present a universal lipoid granulomatosis involving the bones (right humerus, left femur, left ilium, sacrum, lumbar vertebræ 2-v, coccyx), dura, pleura, epicardium, Glisson's capsule, the pancreas, fat tissue surrounding the adrenals, the kidneys, aorta, urinary bladder, testes, mesentary and omentum.

Samples of the lipomatous lesions contained a high percentage of cholesterol, while the ordinary fat deposits contained pure triglycerides (neutral fat).

The brain (Dr. Globus): The calvarium is thickened. The dura is moderately adherent throughout. On removing the skullcap a yellowish deposit is noted over the dura, along the course of the branches of the right middle meningeal artery, and along the anterior portion of the superior sagittal sinus. The same yellow deposit is present on the adjacent areas of the inner table of the skullcap. The left lateral venous sinus is practically obliterated by the orange brown tissue.

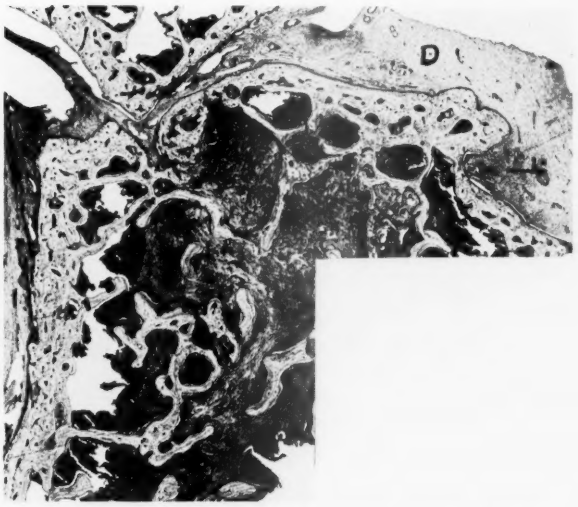

Fig. 3. Section through the apex of the petrous pyramid showing diploic spaces almost entirely replaced by lipoid granulation tissue. The adjacent overlying dura is similarly infiltrated by this tissue. *D*, represents Dura. *L*, Lipoid Granulation Tissue. (Hemalum and Sudan Stain.)

The superior sagittal straight and cavernous sinuses as well as the torcular herophili are partially infiltrated by the yellowish brown tissue, reducing the cross section of the venous channels to a marked degree. There are a few yellowish deposits and calcific plaques along the falx cerebric. The vessels at the base show moderate atherosclerosis. The pituitary is smaller than normal.

Diagnosis: Cerebral arteriosclerosis, chronic adherent pachymeningitis, lipogranulomatosis of the skull, dura and venous sinuses. Microscopic sections of the cerebrum, stained with hematoxylin and eosin, reveal normal architecture and but very slight mononuclear infiltration of the meninges and around a few blood vessels. Some vessels show hyaline changes. Sections taken through the left lateral sinus show the dura infiltrated by whorles, rosettes and strands of fibrous connective tissue, richly cellular, containing numerous fat cells. There are present numerous multinucleated giant cells with eccentric nuclei and foamy cytoplasm. Fat stains of these sections reveal that these infiltrating cells are rich in fat content. The pituitary contains an increased amount of basophilic cells, some of them vacuolated.

Temporal bones.—The dura over both petrous pyramids show yellowish infiltrations. The left sigmoid sinus is almost entirely ob-

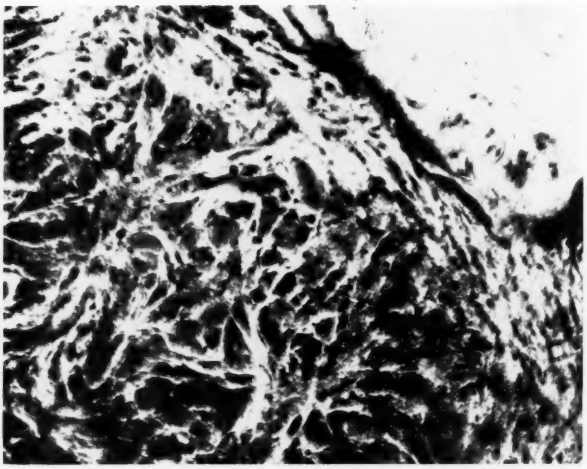

Fig. 4. Higher magnification of lipoid granulation tissue showing lipoid containing cells.

literated by orange brown infiltration. The right mastoid process on section is markedly dense and sclerotic. There are practically no pneumatic structures present.

Histologic examination of the left temporal bone: Except for small segments at various levels removed for study of frozen sections the temporal bone was sectioned serially. Sections were stained with hemalaum and eosin and Sudan. They were also studied with the aid of polarized light. The tympanic membrane was markedly thickened and scarred and has a large central perforation. The epidermis of the tympanic membrane has grown around the margins of the perforation and has partially lined the inner tympanic wall, to form a cholesteatomatous matrix (Fig 1). The tympanic cavity is traversed by fibrous strands and contains a small amount of purulent exudate. The mastoid process is markedly sclerotic throughout, little or no pneumatic spaces bing present.

The marrow spaces throughout the temporal bone, particularly in the petrous apex, are diffusely infiltrated with lipoid granulation tissue. The dura likewise shows diffusely scattered infiltrations of lipoid granulation tissue, few normal spaces remaining (Figs, 2, 3, 4). To a great extent this granulation tissue has undergone fibrosis, which is evidence of its strong tendency toward healing. Very large elongated and spindle shaped cells containing lipoid substance are

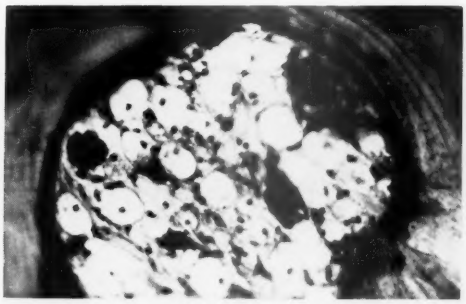

Fig. 5. Large round and oval cells laden with lipoid within the bone marrow (also known as Xanthoma or Foam cells).

diffusely scattered throughout the tissue. This is particularly brought out by the Sudan stain, which stains the lipoid substance pinkish red. In other areas the lipoid granulation tissue does not show such advanced changes. While the vast majority of the lipoid containing cells are elongated, some are round or oval shaped (Fig. 5). These are particularly prevalent in the more recent lesions. A relatively small amount of the neutral fat is also present in the bone marrow. Many of the central canals of the Haversian system likewise show the lipoid granulation tissue. There is evidence of new bone formation but without bone resorption about the margins of the marrow spaces. With polarized light double refractile crystals (cholesterin) are observed both within and without the cells.

COMMENT

This case presents unusual clinical and pathologic features which are worthy of mention. The age of the patient is unusual. Hand-Schüller-Christian's disease is by far more prevalent in children than in adults. Relatively few instances in the latter have been reported. Chester's two cases are examples.

The complete triad of symptoms associated with this condition was lacking in this case. Only one of these, diabetes insipidus, which had become evident nine years ago, was present. In this respect it resembles the case of Thompson, Keegen and Dunn, in which the symptoms of diabetes were the first and only ones for a period of two and a half years. The defects in the membranous bones, the second of the triad, was not detected until shortly before death, though repeated x-ray examinations of the skull were made. The character of the lesions (lipoid granulations) in the temporal bones, however, indicated that they had existed for a considerable length of time.

The history of repeated attacks of middle ear suppuration with increasing deafness, over a period of seven years, is of interest. Naturally the question arises whether these attacks were of specific nature secondary to the Hand-Schüller-Christian disease or whether they were the usual intercurrent infections. Histologic examination offered no conclusive evidence for solution. Though characteristic changes, i. e., lipoid granulations, were observed in the various parts of the temporal bone, the middle ear showed none of these. The picture here was rather that of the usual chronic middle ear suppuration with cholesteatoma. It is possible, however, that the more typical changes were also present in the tympanum at some time in the course of the illness but that they had healed in the interim. As is well known and as was demonstrated in the histologic sections, the lipoid granulations in the flat bones and elsewhere showed strong tendency toward spontaneous healing.

The lesion in the dura, compressing and partially obliterating the lumina of various venous sinuses, is a rare feature. Only in Greifenstein's report of Dietrich's case was a similar condition noted.

From the foregoing observations the deduction can be made that the diffuse involvement of the temporal bone (mastoid, petrosa and apex) was in all probability part of a generalized distribution of the lipoid granulomatosis.

SUMMARY AND CONCLUSIONS

A brief description of Hand-Schüller-Christian's disease is given, emphasis being placed on the aural manifestations. A case is reported for the first time, to our knowledge, in which histologic study in serial section of the temporal bone was made. The histopathologic changes in the temporal bone are, in a general way, similar to those observed in other flat bones of the body. The clinical manifestations in the ear, as comprised from various reports of the literature, are classified. Any or all of the component parts of the temporal bone may be involved, namely, the external ear, middle ear, mastoid, petrosa, etc. When the region of the mastoid is the site of the lesion it must be differentiated from a mastoiditis, Roentgen examination is of value in the differential diagnosis.

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LXIX

RELATIONSHIP OF CLIMATE TO DISEASES OF THE UPPER RESPIRATORY TRACT*

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PHYSIOLOGY OF THE UPPER RESPIRATORY TRACT.

Huntington, in his work entitled "World Power and Evolution," emphasizes man's dependence on climate, and avers that it has been the most important factor in his evolution. The human body is dependent upon atmospheric environment and, by the respiratory apparatus, it adapts itself to variations of temperature and humidity. Nature in its wisdom has fashioned the body so that it is particularly susceptible to the influence of the agent or medium to which it is adapted. Therefore, it is only reasonable to assume that the respiratory tract is sensitive to abnormal atmospheric conditions.

Many centuries ago Hippocrates wrote after this fashion:

"Whosoever wishes to investigate medicine properly, should proceed thus. In the first place, consider the seasons of the year, and what effect each of them produces, for they are not all alike, but differ from themselves in regard to their changes; then the winds, the heat, the cold, such as are common to all countries and such as are peculiar to each locality."

We consider first the physiology of the nose, whose function is, as a respiratory organ, to heat, moisten and filter the air and to act as a protector against the inspiration of bacteria. In the Polar regions it warms the air to such an extent that it removes the danger of freezing lung tissue. It gives off moisture at the rate of six ounces in twenty-four hours, which acts as a protective film over the mucous membrane and helps maintain water balance in the body. The nose also acts as a filter by removing dust from the inspired air and secreting a thin, albuminous liquid which serves as a protection against the entrance of bacteria to the nasal mucosa. This heat and moisture mechanism of the body is controlled by the vasomotor system of the upper respiratory tract and the skin.

*Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, 1936.

Heat dissipated through the skin (approximately 80 per cent) is given up by evaporation, convexion and radiation. Heat given up by the nasal mucous membrane (approximately 20 per cent) is largely by radiation. In this way the skin regulates the internal temperature and keeps the surface of the body warm, while the nasal mucous membrane serves the function of warming the air for the respiratory organs.

For the patient who lives in a cold, damp atmosphere, such a process accounts for decreased moisture eliminated by both nose and skin, with a resulting nasal stasis; while the patient who lives in warm, dry air, loses moisture by the skin to such an extent that an insufficient supply of moisture exists in the nose to make it function properly, and nasal dryness is the result.

Warm, moist air (about 50 per cent humidity) would result in the nasal mucosa being properly moistened, and at the same time enough moisture would be eliminated by the skin to keep the water balance normal and avoid nasal stasis.

Thus, we can see how low winter temperature with accompanying high humidity, predisposes to infections of the upper respiratory tract more easily than high summer temperature with low humidity. When the body is exposed to air, either damp or dry, of a temperature and humidity that produces a loss of heat from the body faster than the heat mechanism is able to produce heat within the body, a chill results. This heat production mechanism, the writer believes, is affected by the presence of infection, i. e., an infected person will not produce body heat to such a great extent, due to subnormal metabolism, as a healthy person and is, therefore, susceptible to slighter variations of temperature. Also, it is noticeable that a higher temperature in which to live comfortably is required of the older than the younger generation.

Dry air, as studied by the Department of Public Health in Chicago,[1] causes irritation in the mucous membrane, sleeplessness, irritability, fast pulse and dryness of skin. Chronic bronchitis is aggravated by irritation. Dry air also causes burning of the eyes, due to rapid evaporation of secretions of lining membranes.

Too much humidity interferes with proper evaporation and dissipation of body heat, thus accounting for a great prostration, as when Chicago was reported to have a temperature of only 88 degrees about July 17, 1934, with humidity of 88 per cent, which resulted in many heat prostrations. At the same time San Bernardino, Cal., and Phoenix, Ariz., frequently showed temperatures from ten to fifteen de-

grees higher with low humidity of around 30 per cent, and with few if any heat prostrations. Thus, we are convinced that excessive moisture makes hot air feel hotter and cold air feel colder.

Consequently, air which is too moist for its accompanying temperature is unhealthful. Many writers attribute the same effect to very dry air, but this writer believes this statement should be qualified to apply to the condition of air within buildings, while the air on the desert rarely ever contains enough moisture at ordinary temperatures to be prostrating, and becomes intolerable only at low humidity and when the temperature reaches over 110 degrees in the shade.

Mention should be made of the allergic patients who are sensitive to the dust that is frequently present in dry desert air. These sensitive patients find the desert very uncomfortable as a result of this irritant.

In a paper by Dean Blake,[2] he states: "The idea that the thermometer alone registers the heat and cold experienced by the human body is entirely too prevalent, judging by the opinions of health seekers who examine the data in the weather bureau offices. Other climatic elements, such as wind movement, rate of evaporation, and the amount of water vapor in the air also affect our bodily sensations of heat and cold. Severe cold, for instance, becomes unendurable when accompanied by wind, and calm weather with a hot, muggy atmosphere is also productive of great discomfort and even fatalities.

"Moist air has greater capacity for heat than dry air, providing the temperature is the same. At 32° F. moist air 'feels' colder than dry air, because under these conditions it takes away heat from the skin faster and leaves the skin colder. When the temperature is higher the heat is kept in the body by the humid air because, owing to poor conduction and radiation, evaporation is hindered and heat loss is prevented.

"Most writers agree that damp air induces nervous depression, slower blood circulation and quiet sleep, while dry air acts as a nervous excitant and causes sleeplessness and restlessness."

In a paper by Daniel Weaver,[3] he states in part:

"The mucous membrane of the whole upper respiratory tract and lungs secretes about one and one-half pounds of water each twenty-four hours in a normal atmosphere. This is for protection of the delicate epithelium, just as the lacrimal gland secretes a fluid to protect the epithelium of the cornea, without which the cornea would be destroyed in forty-eight hours. If, through external or internal

influences, or both, the moisture is not maintained over the mucous membrane, destruction of the epithelium follows.

"All things that come in contact with the mucous membrane of the nose, whether intentionally or inspired through the air, and which are irritating to the membrane and cause corrosion or exfoliation of the surface epithelium, are potent factors in the production of colds.

"Dust particles in the air, especially those of soluble lime, are exceptionally irritating and cause corrosion. A sudden change in the temperature of the air we breathe, fumes of noxious gases, and excessively dry atmosphere are destructive forces.

"The lime dust of a Sunday afternoon automobile traffic is destructive to the epithelium cells, and the bacterial flora present at all times gain a foothold in the denuded surface—result, a cold. The same is true of irritating dust in sweeping with a broom, or the dust behind the threshing machines and that arising in various dusty vocations. But the most serious source of injury to the epithelial covering of the mucous membrane is drying, due to low humidity of the atmosphere in artificially heated homes, public halls, schools and places of business. The effect is well illustrated in the epidemics of colds among school children when fall heating begins. The upper respiratory tract cannot protect itself, except for a short time, against bacteria or irritating foreign matter in an atmosphere of low humidity. The membrane becomes too dry to function properly as a filter, thermostat or humidifier, and the bacteriocidal function is practically destroyed. The surface epithelium in low humidity is exposed to the destructive influence of dryness and foreign matter in the atmosphere which it cannot resist indefinitely. The destruction is followed by bacterial infection-colds.

"In order that the nose may function normally, the atmosphere indoors should be of a humidity of 50 per cent saturation, and a uniform temperature of 68 to 70 degrees F. Any marked variation from this humidity and temperature is an agent for respiratory infection."

A. Lowndes Yates[1] makes the following two conclusions:

"First, that the severity of a sinusitis, or sinus infection, depends upon the power of the mucous membrane to prevent its destruction by the inspired micro-organisms, and if the property that mucus possesses of preventing the passage of water and hence of water-soluble toxins is destroyed, that a progressively increasing poisoning by these toxins of the ciliated epithelium occurs.

"As the ciliary paralysis increases in amount, the secretion of mucus is also paralyzed until finally the micro-organisms come to dwell in a symbiotic state in the sinuses, and the toxins from them are readily absorbed with the production of remote symptoms.

"Second, that the intimate relation of the tracts from the sinuses to the eustachian tube suggests that eustachian catarrh is a secondary manifestation of sinusitis, and that the intimate relation of the tracts from the sinuses to the tonsil suggests that the initial enlargement of the tonsil is frequently due to sinusitis, although tonsillar enlargement may be due, of course, to other causes."

McDonald, Leisure and Lenneman[5] have pointed out that ciliated epithelium, lining of the sinus, exerts a certain biophysical defense against invasion of pathologic bacteria and that this ciliary action might be augmented or retarded and local resistance raised or lowered. Among the factors augmenting ciliary action was temperature, warming increased and raised resistance, while cooling definitely retarded its action and lowered resistance.

The effect on persons exposed to heat and very low humidity is a form of prostration with considerable dehydration. These patients evidently lose more chloride than they should, since the taking of larger quantities of chlorides (especially sodium chloride) tends to prevent this type of prostration. Saline given intravenously proves to be the best means of restoring them. These facts suggest a corresponding opposite condition where insufficient sweating or loss of fluid by the skin occurs and prompts us to ask if a lessened sodium chloride intake might be indicated.

Ullman[6] discusses the relationship between chlorides and calcium.

Mention should be made of the variation in sensitivity of patients, i. e., at one extreme we find in atrophic rhinitis the hyposensitive patient who will permit, without discomfort, the placing of applicators, tampons or eustachian catheters. At the other extreme there is the hypersensitive rhinitic who experiences great discomfort from the smallest amount of mechanical manipulation. Thus, we understand why one person may go in the ocean or through a dusty desert or through pollen laden air without much ill effect, while the hypersensitive patient develops a severe rhinitis by doing so.

EFFECT OF CLIMATE ON THE INCIDENCE OF HEAD COLDS.

The Pickett-Thomson Research Laboratory of London conducted an extensive review of over 2,000 research papers on the common cold. The authors who themselves have done careful research on the

common cold for fifteen years, do not hesitate to say that the incidence of the common cold is almost entirely a matter of climate.

On the other hand, W. H. Barrow,[7] in the study of the incidence of colds among students at universities located in different parts of the United States, found that at Stanford University in California, where the climate is mild, practically the same number of students for each hundred investigated suffered from acute colds as did those at Harvard University in Massachusetts.

Townsend,[8] of the United States Public Health Service, conducted a study of a large group, mostly of students of colleges, and of another group composed of medical officers of the Army, Navy and Public Health Service and members of university faculties. The universities were scattered as far as Boston, New Orleans, Chicago, Tucson, Salt Lake City and Berkeley, and the reports covered a period of more than eighteen months. The incidence of colds at the ten universities was found to be remarkably uniform. In fact, the uniformity of attack rate was one of the most significant facts brought out by this study.

On the question of humidity in relation to respiratory diseases, R. M. Hutton,[9] Senior Investigator, Division of Industrial Hygiene, Ontario Department of Health, states in part:

"Comparison between cotton and woolen industry is of interest. The spinning, doubling and weaving of cotton are carried on in conditions of artificial heat and moisture, whereas the similar processes of wool are not. The mortality from respiratory disease among the cotton workers is well above the average, while that among the woolen workers is well below. From bronchitis the cotton spinners suffer a mortality nearly three times as great as the wool spinners; the doublers, about two and one-half times as high, and the weavers nearly twice as high. A similar analysis in cotton weaving alone shows that the mortality from diseases of the respiratory system (bronchitis and pneumonia) of weavers in wet sheds is something like three times that of the weavers in the dry sheds. In these cases, therefore, it looks as though artificial heat and humidity had a causative relation to respiratory mortality."

In a paper by Hilda M. Woods,[10] Division of Epidemiology and Vital Statistics, London, the question of relationship between the unfavorable climatic conditions and the death rate from respiratory diseases is discussed and the seasonal curve of mortality is observed. The winter rise in mortality from respiratory diseases corresponds to the fall in temperature, and with the rise in temperature in the

spring there is a decline in the respiratory death rate. But the month of maximum mortality is not in the depth of winter but in February —the time of greater variation.

This corresponds with the experience of numerous physicians practicing in the neighborhood of the Great Lakes. Here respiratory infections occur in the late autumn and early winter, decrease through steady cold weather or midwinter, and again recur to a peak during the months of March and early April. The work of many physicians during these "peaks" would show two or three times the number of visits and treatments to patients suffering from respiratory diseases than at any other time of the year. One should note the climatic combination of humidity and marked changes in temperature, i. e., between midnight and midday. During March and November the combination of rain, muddy streets and roads, freezing nights and thawing days, seems to be about the most potent factor predisposing to colds in the writer's experience.

In a study by Russel of two districts of two metropolitan burroughs, Islington and St. Pancras, over a period of twenty-one years, he concluded that fog itself has no appreciable effect on the death rate unless associated with a low temperature and frost, and the effect on the mortality of young children is not so great. He illustrates this by selecting two periods of great fog (1883-84 and 1892-93). In the former period there was no obvious increase in mortality after the fogs. In the latter period there was a large increase. However, in the former, there was no associated sharp fall of temperature, as occurred in 1892-93. The conclusion is that it is essential to bear in mind that, in any study of the influence of weather on the prevalence of disease, the assignment of the increased mortality to any particular factor is not so simple as is generally believed. The fact, often overlooked, is that there are a multiplicity of causes, all of which are highly interrelated, and it is the cumulative effect of these and not the influence of an individual factor that finds expression in an increased death rate from respiratory disease.

Frederick N. Sperry[11] mentions the fact that Government statistics would indicate that the mortality in respiratory diseases, especially influenza, is greater where there is great humidity and severe cold. Moderately cool and relatively dry weather has been marked by lessening colds. The geographical studies of Elsworth Huntington of Yale are at some apparent variance with these statistics. His studies, however, have more reference to civilization and general health. The hardening effect of cold has been noted in Arctic ex-

plorers and in woodsmen who tramp in the snow and are frequently wet and fatigued, yet are free from colds. The records of the Metropolitan Life Insurance Company show that in winter indoor, sedentary workers have nine times as many colds as taxi drivers and four times as many as soldiers in the United States Army.

It is also necessary to consider the sun as a climatic factor. V. S. Cheney,[12] Medical Director, Armour & Co., Chicago, says in part regarding this subject: "During the winter we lose a part of the actinic effect of the ultra-violet rays, due to the sun's southern declination, which requires the rays to pass through a thicker stratum of atmosphere which, especially if smoke-laden, acts as a filter to the rays and materially decreases their chemical action upon the blood, chiefly influencing its calcium content or calcium fixation." As proof of this fact, we point out that skin infections, such as impetigo, which flourish in the winter, will clear up in summer.

Gilchrist and Wilson,[13] writing on "Staphylococcic Infections in Diabetes Mellitus," say that in their observations for the past five years they have brought out the fact that in a large number of diabetic patients they were dealing with a staphylococcic infection of the upper respiratory tract. Their conclusion is that focal infection inhibits the normal production of insulin and that the condition is made worse by damp weather.

CLIMATE AND NATURAL HISTORY

Until the advent of modern bacteriology, climate has always been regarded as a potent factor in the causation of colds. However, since the discoveries of Pasteur, the tendency has been to relegate to the background the old thought and our inclination to over-emphasize one particular factor has caused us to forget many other important points, of which climate is only one. We have thought, in the past, that inasmuch as a draft is only fresh air it is impossible to derive any ill effects therefrom, and overlook the fact that this overdose of fresh air has the ability to abstract more heat from the body than it can afford to lose, thus resulting in a chilling effect, especially if the body is at rest, when heat production is at its lowest point. If we choose to prove these points by experiments we might refer to Lode,[14] who infected his animals with pneumococcus and shaved one-half of the body and, after a warm bath, exposed them to an open window. The result was infiltration of the lungs, sometimes fatally. Jezerski's[14] experiments differed somewhat in that they were milder and more nearly reached real experiences. He did not use such severe cooling methods and did not withdraw nature's method of protection

by shaving the animals. He found that by cooling an already moist, uncovered part of the chest in the case of rabbits or goats, there always occurred some changes in the lungs from simple engorgement to more marked disturbance.

Slight changes from moderate to lower temperature appear to have a stimulating effect on metabolism, while too great or too sudden changes produce chilling, which results in lowered resistance and the invasion of organisms, resulting in the onset of colds, and other respiratory diseases.

Temperature rises ranging from moderate to severe, coming on suddenly, appear to be enervating, lowering resistance considerably and in some patients permitting the light-up of infections that are otherwise arrested. Thus, southeast winds of the Hawaiian Islands and the warm, wet seasons along the Zambesi bring on the dreaded tropical fevers.

In the temperate zones most domestic and wild animals provide for the onset of winter with more elaborate hair or fur and large quantities of fat. The bear, for instance, prepares to hibernate by growing a very furry covering and gaining weight. Mention should be made that fur bearing animals are always marketed in winter when the fur is in its finest condition. Even man in cold climates has a tendency to gain weight up to the onset of winter and usually maintains this extra weight until spring, illustrating the stimulating effect of cold on metabolism.

Among migratory birds the wild goose of the North migrates far to the South when winter comes and remains there until Spring. One can understand why it flies South, but why does it return to the North in the spring? Wild life is concerned with food, cover and climate. These necessities can be obtained in many climates. Why does all migratory bird life move North when Spring comes? Why does the barren land caribou make such long migrations? Why does the salmon cross the Atlantic? Why does the eel travel from the West Indies to the shores of Northern Europe? If we study these natural history problems I believe we will be obliged to conclude that climate, and principally temperature, modify metabolism so greatly that instinctively all wild life is affected by it. The largest bear lives in Alaska. The largest moose lives in the Yukon. The largest tigers do not come from India but from the rugged, mountainous district of Western China. The largest of elephants, the African elephant, seeks the highest plateaus in the mountains separating British East Africa from the Belgian Congo, at an elevation of 7,000 feet. The greatest ape, the gorilla, makes his home in this district.

Similarly, we might trace the wanderings of the human family, and be able to show that they have preferred to seek climates where marked seasonal changes occur, in other words, where they have enjoyed the most physical benefit. History reveals the fact that most of the world's conquering races have lived in temperate rather than torrid zones.

The effect of climate on the Canadian cavalry forces sent to South Africa during the Boer War, about 1900, namely, Lord Strathcona's Horse, is of interest. An officer on his return told the writer that these animals stood the change of climate very poorly, notwithstanding the fact that they had been range horses, or bronchos, and were considered very hardly animals in their Western Canadian home. He stated that six months after they arrived on Orange Free State and the Transvaal scarcely a single head remained alive. This was attributed to change of climate, food and water.

A similar example is the effect on the Eskimo when removed from the Arctic Circle, as experiments carried out by both United States and Canadian governments have shown. Of course, immunity and the effects of civilization are also factors as well as climate. The same applies to officers of the Britsh and American Army and Naval Service in the Tropics. In some instances four years is the maximum of their tolerance.

The writer has observed over a period of fourteen years that hemoglobin percentages, in the hospitals in which he works in Southern California, seem to run uniformly lower than in the North. Tonsil operations require more careful attention. In the writer's opinion, hemostatic pressure after the operation is not enough in many instances, but more care must be taken to ligate bleeders than is the custom in the North. This applies more particularly to adults.

Dr. Clarence A. Mills,[15] professor of experimental medicine, University of Cincinnati, states in part:

"Moist heat that depresses bodily metabolism makes for an increased susceptibility to infection and lessens ability to fight bacterial invasion, once they have gained a foothold.

"I have found definite differences in this respect between the more active people of the North and the Southerners of more sluggish metabolism. Of each 100 cases of acute appendicitis handled in hospitals, the fatality is almost three times as high in the South, with a steady lowering of the fatality rate as one goes northward from the Gulf region.

CLIMATE AND TUBERCULOSIS CASES

No disease of the respiratory system has been more obstinate to treat than pulmonary tuberculosis. Medicine has long since proved worthless and treatment was given up as being useless for a time, until the advent of rest, open air and diet. The writer has for years been interested in the effect of climate on these patients and has observed that at the onset it was customary to send patients to the Pacific Southwest, probably on account of lower humidity, less extremes in outdoor temperature and more hours of sunshine. Undoubtedly, like many other new ideas, climate was overstressed and many fantastic reports were made. The writer has tried to get the truth on this particular subject and has been told by several eminent chest specialists that climate does not amount to more than 10 per cent as a therapeutic measure.

There are features about this subject which have proved interesting. First, why do advanced cases of this disease, when arrested in these Pacific Southwest climates, have to remain there? Experiments have been made on average healthy Eastern students coming to Colorado Springs. Complete blood counts have been done on their arrival and again at the end of three months when they departed for the East. These blood counts and hemoglobin estimations showed uniformly marked increase in cell count and percentage of hemoglobin over control cases which had not visited Colorado Springs but had remained in Eastern cities. Again, these patients whose cases have been arrested, and who remained where they were cured, remain arrested a much longer period of time than those who return to their original locality. Why is this? The writer is not concerned with any particular angle of this subject other than the scientific, and is aware that most tubercular patients are treated at home regardless of climate, and are arrested with average good results. But having visited several centers such as Denver, Colorado Springs, El Paso, Tucson and Phoenix and consulted patients coming from all over the United States and Canada, the writer has been impressed with the uniformity of the personal history of these people, i. e., that nearly all made more rapid progress in the Pacific Southwest, and those that had come too late usually expressed their regret.

V. O. Knudsen,[16] of the University of California, writes as follows:

"The results of an investigation on the absorption of sound in air, published by the author two years ago, indicated that at least in the audible range of frequencies, the absorption co-efficient (1) was from

ten to twenty-five times greater than the co-efficient calculated by classical theory, and (2) increased rather markedly as the humidity of the air decreased. The more recent work of Hopper and of Chrisler and Miller has led to similar results, although their experiments were not designed to determine the absolute values of the absorption co-efficients. In addition, the work of Chrisler and Miller indicates that the absorption increases with a rise of temperature. In regard to the absorption co-efficience of sound in air at 20° C., containing different amounts of water vapor for a tone of 6,000 cycles dying away in a six-foot chamber filled with air at 20° C., it will be seen that the rate of decay of sound was slowest for the most dry air; that it reached a maximum at a relative humidity of about 13 per cent; and that it decreased again for higher humidities. Similar series of decay curves were obtained for frequencies of 500 to 11,000 cycles. In each case the rate of decay was slowest for the dry air; the rate of decay reached a maximum at a certain humidity, between 5 per cent and 20 per cent, and diminished at higher humidities."

In a recent communication to the writer, Professor Knudsen states:

"Normally oxygen molecules are of two types—those which are normal or non-vibrating and those which are in a state of vibration. In general, the number of vibrating or excited oxygen molecules is determined by the temperature of the gas. However, recent experiments which I have conducted on the absorption of sound in oxygen and water vapor indicate that collisions between oxygen molecules and water vapor molecules will result much more frequently in the production of a vibrating molecule than will similar collisions between two oxygen molecules . . . . and further, if water vapor will produce such a prominent effect upon the sound-absorbing properties of a gas, as oxygen, it is not at all impossible that the action of water vapor or oxygen molecules may result in an oxygen gas which has different chemical and physiologic effects from oxygen which is free from water vapor, or from oxygen which contains a different percentage of water vapor."

The following is a practical illustration of the effect of humidity on respiratory diseases. The writer, who lives in a city of over 100,000 population, on the seacoast, where the humidity average during the winter months is approximately 90 per cent for the twenty-four hours, has conferred with pediatrists on the treatment of acute and chronic brinchitis and pneumonia (especially bronchopneumonia). These specialists practice what they call the "closed window treatment." This was found necessary because, while freezing is practi-

cally never known in this city, yet the combination of a temperature as low as 32 degrees F. is quite common in winter, with a humidity, as stated, or close to 90 per cent. To open windows in the sick room and permit a sick child or adult to inhale into a diseased lung this kind of air has proven very detrimental.

Some patients have, therefore, been sent to the desert. Others who have remained at home are treated by this closed window method, i. e., the air is taken into the room and warmed to an appropriate temperature before being inhaled. By so doing, the humidity is lessened and the temperature is raised, without changing the constituent gases. Patients have recovered uniformly well under this treatment.

Also, the treatment of sinusitis during cold, humid weather in this seacoast region resembles this closed window therapy. For instance, the writer has found it very difficult to obtain good results on oil workers who are on night shifts of eight hours, exposed to cold, humid ocean breezes. The same applies to officers and men in the Marine Service, more especially the officers.

Consequently, instead of characterizing the sinus or chest patient as a neurotic or rainbow chaser, the writer believes that through experimentation in the physicist's laboratory will be unraveled the reason why these become better oxygenated, develop better metabolism and have scantier secretions and freer drainage in certain climates. To this should be added the therapeutic value of sunshine and winds, and even if climate does not amount to more than 10 per cent, in serious borderline cases 10 per cent often means the difference between success and failure.

Table I.—Group 1 shows a group of cities which have low temperature, high humidity and few hours of sunshine. Group 2 shows two cities of Florida, which have high temperature, high humidity and a high percentage of hours of sunshine. Group 3 of this same table shows high temperature, low humidity and the greatest number of hours of sunshine.

Table II.—It will be observed that from Group 1 the replies are practically all in the affirmative. From Group 2 and Group 3 the replies are all negative. In other words, out of twelve replies, eleven of the first group are in the affirmative; and out of the twelve replies in the second and third groups the entire twelve are negative.

A study of the two following tables would indicate that climate is a definite factor in the etiology of sinusitis.

TABLE I.—METEOROLOGICAL RESEARCH (METEOROLOGICAL SURVEYS)

	Temp. (12 Mo.)			Rela. Hum. (12 Mo.)			Sunshine	
	Maximum Temp.	Minimum Temp.	Average Temp.	Maximum Hum.	Minimum Hum.	Average Hum.	Monthly Aver. Hrs. Sunshine	Percentage of Possible Hrs. of Sunshine
GROUP No. 1—								
Vancouver*	.....	.....	49			80.41	152.75	.....
Seattle**	80	37	58.5			80	152	.....
Toronto	70.1	22.8	46.45			69	172.36	46
Winnipeg	70.09	—7.4	37.9			77	173.5	.....
GROUP No. 2—								
Miami Beach	83.8	62.8	73.3	78	74	76	249	67
Palm Beach	94	45	74.5	78	74	76	249	67
GROUP No. 3—								
El Paso	81.7	44.8	63.25	54	27	40.5	.....	90.70
Phoenix	103	38.5	70.75	56	28	42	.....	84
Tucson	107	20	63.5	54.4	22.2	38.3	.....	80

**Seattle (No relative humidity record given, but its proximity to Vancouver would lead us to assume that the relative humidity would compare with that of Vancouver).

TABLE II.—QUESTIONNAIRE TO DOCTORS

Question: Has your climate a tendency to produce chronic sinusitis which does not respond readily to treatment?

Group No. 1

Vancouver	Yes (1 reply)
Seattle	Yes (3 replies)
Portland	Yes (2 replies); No (1 reply)
Winnipeg	Yes (1 reply)
Toronto	Yes (4 replies)

Group No. 2

Tampa	No (1 reply)
Palm Beach	No (1 reply)
El Paso	No (3 replies)

Group No. 3

Phoenix	No (1 reply)
Tucson	No (1 reply)
Denver	No (3 replies)
Philadelphia	No (2 replies)

Following are excerpts from letters sent to the writer from doctors practicing in the above-mentioned cities, which bear out the information revealed in the two preceding charts.

In reply to the question:

Has your climate a tendency to produce chronic sinusitis which does not respond readily to treatment?

Vancouver, B. C.: "Yes, in my opinion our coast climate does play a very important part in the causation of sinus infection and also in prolonging treatment. Especially in our winter season we have quite a quantity of foggy weather and the atmosphere is generally damp. I notice that quite a number of sinus cases (mild cases of ethmoiditis) improve markedly when sent inland to the dryer climate. There is no doubt that most chronic cases of sinusitis require an operation, as there is always an anatomic abnormality present. I do not know how this compares with other locations, but having spent two and one-half years in New York, I must say I cannot see much difference between the two. These places, of course, are both foggy and situated near the water."

Seattle: "Temperature and humidity are probably factors in sinusitis, but I believe that sunshine is a very helpful agent, for this winter (1933-34), we have had more sunshine than I ever remember and far less sinusitis. In areas on this Puget Sound country where there is a great deal of wind there seems to be less trouble than where we have little stirring up of the atmosphere."

Florida: "I do not think we see the percentage of chronic sinusitis that is seen elsewhere (that is, in northern climate), due to the fact that the common cold is not so prevalent, and clears up more rapidly in a mild climate. Of course we see a number of chronic sinuses; however, the percentage is greater among those that have come from the North."

Tucson: "Would add that I have noticed, since being in this locality for fifteen years, that there has been only an extremely small number of patients, almost negligible, in my practice, residing here, who have been afflicted with sinusitis which has become chronic. However, I must say that I believe I note almost as much acute sinusitis as in more humid climates where I had formerly practiced."

Denver: "If our climate has any effect it is deterrent rather than productive of chronic sinusitis. Lack of humidity and neglect of nose, with resultant dryness appears to be a factor in many cases. Many cases improve to such an extent that they do not visit the

doctor on arriving in Denver, and information to this effect is frequently gained incident to history of other conditions. Improvement seems to be more marked for a time in individuals from other climates than in the case of Colorado natives."

It should be taken into consideration that the cities mentioned in the Pacific Southwest have a large tubercular population and, according to Hajek,[17] "it is truly remarkable, as shown by E. Frankel Wertheim, that nearly every third tuberculous body (evidently autopsies) was found with an inflammatory affection of the accessory sinuses. These inflammatory affections were not at all different from those occurring in the other diseases; they did not depend upon tuberculous infections, since a true tuberculous infection of the mucous membrane of the accessory sinuses is rarely seen."

SUMMARY

1. That "just as one swallow does not make a summer," neither does one factor constitute an entire etiology. In this paper the writer deals with one cause only, namely, climate, and acknowledges the importance of all other factors.

2. That the nose is a factor in maintaining the water balance of the body, and the efficiency of its ciliary and other functions definitely depends upon changes in climate.

3. That the incidence of head colds, while occurring about the same time of year over the entire continent and Europe, seems to be definitely related to changes in humidity and more especially to marked changes of temperature.

4. That a study of natural history suggests that the metabolic condition of all life, both wild and civilized, is affected by climatic changes.

5. That patients suffering from tuberculous disease of the upper respiratory tract appear to do better in certain climates.

6. That climate is a distinct factor in the etiology of diseases of the upper respiratory tract and consequently climate must be considered a factor in its treatment.

LONG BEACH, CAL.

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WHEN IS SINUSITIS CHRONIC?

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There are few terms in medicine used with less discrimination than "chronic sinusitis." And this but reflects an unfortunate, but widespread, vagueness or looseness of definition. What is and what is not "chronic" often appears to be simply a matter of viewpoint. Yet, to have meaning, this diagnosis must be used with the same care when describing sinus pathology as when applied to tissue damage elsewhere in the body.

According to various opinions, this disease can be cured in divers and sundry ways. Methods recommended cover the entire gamut from topical applications and vaccines, through minor surgery, to actual removal of the affected mucosa. In each category the percentage of good results is high but the proportion of "cures" is most attractive in those series receiving some form of "treatment" or only minor surgery. One is almost persuaded at times that chronic sinusitis is one of the easiest of all diseases to eradicate. A more reasonable conclusion, however, is that fundamental differences in pathology account for what is obviously confusion.

Undoubtedly there is wide lack of agreement as to just what evidence warrants a clinical diagnosis of chronic disease. Yet this need not be the case, and probably is not, among careful workers. Adherence to any of several methods of investigation will result in reasonable accuracy and fair agreement.

Histologically, a diagnosis of chronic sinusitis requires definite structural changes throughout the mucosa, some of which are essentially permanent and irreversible. Such tissue alteration is the result of infection through progressive vascular damage. Cellular infiltration, fibrosis and hyalinization are as marked in the blood vessel walls as in any part of the mucosa, and the resulting tissue changes are of far reaching importance.[1]

Grossly, the outstanding characteristic is increased thickness of the sinus mucosa. This thickening is irregularly distributed and consists of edema, cellular infiltration and what is most important—

hyperplasia. The effect of these changes is to impair, markedly and presumably permanently, local tissue defense against pathogenic bacteria.

Standard descriptions of chronic pathologic changes in sinusitis are definite. From Jackson and Coates:[2]

"The result of long-continued irritation is to produce degeneration of the more highly specialized functioning part of the membrane and its replacement by fibrous tissue. If the irritant is sufficiently low grade the hyperplastic changes may prevail, but if the irritation is more severe there is more tissue destruction and exudation, producing ulceration and granulation tissue with eventually polypoid degeneraiton or fibrous and atrophic changes."

From Skillern:[3]

"Chronic sinus inflammation . . . (1) hyperplastic, (2) ulcerative. Hyperplastic type: The mucous membrane is of grayish color, often wrinkled and papillomatous (and) edematous . . . Connective tissue is thickened . . . Ulcerative type: Not found as such, but is rather a hyperplastic condition associated with ulcerosis."

It is unthinkable that all nose and sinus infection must be considered as either acute or chronic. There is ample use for such terms as subacute, recurrent acute, etc., to describe intermediate conditions.

Careful rhinologists require definite objective evidence before making a diagnosis of chronicity. This caution arises from familiarity with tissue pathology, both gross and microscopic. Yet one sees this diagnosis applied loosely, often on the basis of history and duration of complaint. Too often such evidence as should be the reason for further investigation becomes instead the basis of diagnosis and treatment.

Between these conceptions there is wide and fundamental disparity. Indeed the relative proportion of chronic cases in one's practice may be small or great, depending on the degree of care in diagnosis.

A pertinent quotation from Ballance[4] appeared in an article by Williams[5] in the Journal of the American Medical Association some time ago:

"Want of agreement among serious workers about a question of surgical treatment arises either from the lumping together of clinical conditions having an essentially different pathologic basis or from essential pathologic conditions not being clearly understood. All true

and lasting surgical practice is based upon pathology, and when once the pathology of an affection is clearly appreciated, divergence of view as to its treatment should disappear."

In reporting results of treatment of chronic sinusitis it should first be made evident that the cases are truly chronic. A "cure" should include not only temporary relief from local and general symptoms but evidence of the return of the diseased mucosa to normal, as evidenced by follow-up examination. This would make evident an awareness that quiescent periods between exacerbations are characteristic of chronic infection, whether of the sinuses or of any other body tissues.

It is probably easy to be misled as to what is and what is not true chronic disease. A review of my own cases for the past three years is illustrative of this. I am considering only those patients in whom chronic sinus disease might be assumed to be present on the evidence obtained in routine history and examination. In a small percentage chronicity is obvious. In a few others a complete survey may be done at once. However, in most instances, the final diagnosis must be delayed in the interest of honesty and accuracy. This is because patients commonly seek aid during an acute process. It is seldom justifiable to assume chronicity at such a time, no matter what the history. It is my practice to make a later examination of such cases and every effort is made to do this not earlier than one month after subsidence of all evidence of acute infection. This allows time for resolution.

The delayed examination, or sinus survey, includes radiographs after instillation of iodized oil as a contrast medium. The oil is easily and painlessly instilled by lateral head-low posture[6] and the Proetz[7] displacement technic, or if that is not possible the maxillary sinus may be filled by direct injection. Displacement is much to be preferred, as by it one can usually reach most or all of the sinuses on one or both sides. The radiographs are taken at once and include straight postero-anterior and straight lateral views in the erect position. The purpose is, of course, to visualize the thickness of the sinus mucosa.

More than 75 per cent of such patients have failed to show any appreciable mucosal thickening within the sinuses. Included are quite a number who had been advised elsewhere to have radical antrum surgery for removal of chronically diseased mucosa. The remainder usually showed sufficient hyperplasia to leave no reason to doubt chronicity.

In the investigation of sinuses this method has been widely used ever since its introduction by Proetz[7] ten years ago. Yet it is not presumed that it is the last word in diagnosis, that it answers all questions and purposes, or that it precludes other or further examination. It is believed, however, that it is based on rational premises and that it is reasonably accurate. It has the advantages of simplicity and directness, and in practice has seemed satisfactory. One can easily anticipate a certain amount of criticism of this or any other method of diagnosis but not of its purposes.

Diagnosis must, of course, take into account the occasional case with only slight mucosal thickening but undoubted chronicity. Also, one must recognize the presence and effect of allergy and give it intelligent attention in the interpretation of the whole case and in the plan of treatment. The same is true of endocrine disorders and of any other factors bearing on the case.

Admittedly it would be pointless to report improvement or cure in cases where no hyperplasia is demonstrable. It is common experience that these patients usually secure satisfactory relief by treatment, regardless of the duration of symptoms. I prefer to think of them as subacute or recurrent acute. In many instances the condition is based on lack of understanding of the first principles of nasal hygiene. But unless due care is exercised in diagnosis they can form the major portion of a "series" of "chronic" cases. The significance of this is that their inclusion adds luster to any regime that improves nasal ventilation.

To often optimistic reports of treatment of chronic sinusitis appear in periodicals for the general medical audience rather than in journals devoted to problems within our specialty. No doubt this lends an air of verification and effectively prevents adequate criticism.

SUMMARY

Topical and other methods of treatment are constantly before us that reputedly cure all but an insignificant percentage of cases of chronic sinus disease. Some methods are elaborate and costly. It seems justifiable to wonder if the same optimism would exist if more critical attention were allotted to diagnosis.

The clinical diagnosis is one's prediction of the histopathologic picture. It is important to know the premise upon which a diagnosis is considered justified. Otherwise there is no basis upon which the value of work can be measured or accurately checked. And only thus

can one avoid the suspicion of "lumping together clinical conditions having an essentially different pathologic basis."

A reliable criterion of sinus pathology is the ability or lack of ability of sinus mucosa to recover following acute inflammation. For a case of sinusitis to be cured—not to say improved—by a relatively simple procedure is good presumptive evidence that the pathology had not become chronic but had been simply acute or subacute.

It is my experience that of the run of cases that on routine examination appear to be chronic, the great majority cannot justifiably be listed as such. More than 75 per cent fail to show hyperplasia after a reasonable delay to allow resolution to occur following acute congestion. This high proportion parallels roughly the published percentages of "cures" of "chronic sinusitis" by topical methods of treatment and by minor surgery.

MEDICAL BUILDING.

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LI

OSTEOMYELITIS OF THE FRONTAL BONE*

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BOISE, IDAHO

In going through the literature in the preparation of this paper I found that practically all the men writing on this subject are mostly in accord, and that nearly all the articles started out with "Osteomyelitis of the frontal bone is a progressive disease and its surgery must be timely and radical." It is also recognized that there are two types of osteomyelitis; one the spontaneous occurring without previous operative intervention, and the other the postoperative type. Three of my cases have been spontaneous and one postoperative.

The title of my paper is "Osteomyelitis of the Frontal Bone," but in my group of cases I found two that were not of the frontal bone but that were of such marked interest that I decided to report these. I feel that we should first consider the anatomy that makes it possible for us to have this type of infection. The following material on veins of the diploe and the emissary veins is abstracted from "Gray's Anatomy of the Human Body:"[1]

> The diploic spaces of the cranial bones in the adult contain a number of tortuous diploic canals, surrounded by a more or less complete layer of osseous tissue. The veins they contain are large, their walls being thin and formed only of endothelium resting on a layer of elastic tissue; they present at irregular intervals pouch-like dilatations which serve as reservoirs for the blood.
>
> So long as the cranial bones are distinct and separable, these veins are confined to the particular bones; but in old age, when the sutures are united, they communicate with one another and increase in size. They communicate in the interior of the cranium with the veins and sinuses of the dura, and on the exterior of the skull with the veins of the pericranium. They consist of (1) the frontal diploic vein, which opens into the supraorbital veins by an aperture in the supraorbital notch; (2) the anterior temporal diploic vein, which is confined chiefly to the frontal bone, and opens into one of the deep temporal veins through an aperture in the greater wing of the sphenoid; (3) the posterior temporal vein, which is situated in the parietal bone, and terminates in the lateral sinus through an aperture at the postero-inferior angle of the parietal bone or through the mastoid foramen; and (4) the occipital diploic vein, the largest of the four, which is confined to the occipital bone, and opens into the lateral sinus or the torcular herophili.

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The emissary veins pass through apertures in the cranial wall and establish communications between the sinuses inside the skull and the diploic veins in the diploe, and the veins external to the skull.

ETIOLOGY

There are many factors contributing to the etiology of osteomyelitis. The virulence of the organism and the resistance or immunity of the patient to the particular organism are the two principal factors involved. The etiology of spontaneous osteomyelitis is even more obscure than the postoperative type. Many times it follows trauma. The most common organism is the staphylococcus pyogenes aureus.

Most of the cases reported in the literature are the postoperative type, and there does not seem to be any unanimity of opinion as to why osteomyelitis of the frontal bone should develop following an operation on the antrum. There are more cases of osteomyelitis reported developing following the intrasanal antrum operation than following the more radical Caldwell-Luc.

Dan McKenzie[2] states: "A minor interference is more likely to leave behind it active suppurating foci in contact with the wound." He further states that "we know that osteomyelitis may follow such simple and restricted proceedings as the intranasal antrum operation, the intranasal frontal sinus operation, and even the curetting of an ethmoid for polypi. Indeed, if the reports represent the usual run of cases, the disease is more frequent after small than after large interventions."

In the spontaneous variety of osteomyelitis there seems to be less tendency for it to be of the spreading type. The infection and destruction remain more diffuse and there is not the general constitutional reaction that there is in the postoperative type.

Furstenberg[3] does not seem to agree with the two classifications of the disease. He has serious doubts that as such they are truly clinical entities. "On the one hand we have an infection which by reason of its great virulence and because of a poor constitutional resistance on the part of the patient spreads wildly through all of the osseous tissue which lies in its path. In another instance, because of less favorable influence to bacterial invasion, the disease limits itself to a part of the cranium and shows a tendency to spontaneous resolution."

Furstenberg also states that he has the feeling that septic thrombosis of the veins of the mucous membrane of the frontal sinus is the

cause of osteomyelitis of the frontal bone. Mosher[4] has a pathological specimen to prove this.

PATHOLOGY

Adson[5] states concerning the pathology:

"Osteomyelitis of the skull develops when infection has been introduced into the diploic spaces after emissary and diploic veins have become thrombosed. Control of the destructive processes depends on the arterial and venous circulation, the immunity established, the extent to which pressure under the periosteum is relieved by drainage, and removal of necrotic and avascularized bone."

Furstenberg states that one may find in pathologic studies of osteomyelitic lesions innumerable signs of early injury to the inner table of the calvarium. "In fact, it may be correctly said that the inner plate is the first to be destroyed. But what is still of greater significance is the fact that the inner table may be the only part of the bone primarily affected, while the more superficial medullary tissue and external plate may remain uninvolved. In this alone, there is considerable proof of a primary intracranial yet extradural origin of an infection that may subsequently erode and destroy the entire cranial vault."

SYMPTOMS

The spontaneous type of osteomyelitis may have very few symptoms. There is generally a swelling over the involved area. There may be almost no pain or tenderness associated with this swelling, as will be shown later in the report of my cases. The postoperative or fulminating type of osteomyelitis, however, is entirely different. There is generally swelling that extends up to the frontal bone, marked tenderness, early fluctuation, and the patient shows all the signs of being severely ill.

SURGICAL TREATMENT

Only a few writers on this subject advise against the radical type of surgery. Most of them are in accord that adequate drainage is necessary, that all necrotic and white dead bone should be removed until live bleeding bone is exposed. Mosher, in his splendid article, advocates the desirability of going at least an inch above the involved area. He states that once osteomyelitis has set in, the only chance of saving the patient lies in the immediate and entire removal of the diseased bone. The whole thickness of the bone cranium must be removed down to the dura. Further, the superficial area of the bone

resected must exceed the obvious limits of the disease. Intervention must be comprehensive. Resection must be radical. In doubtful area it is safer to remove than leave it, so that the removal should be carried wide of the disease in all cases. The size of the resections of the bony cranium need arouse no apprehension, for after recovery the defect will be made good by osseous regeneration. It is also generally conceded by most operators that the x-ray is not a criteria as to the extent of the osteomyelitis. Mosher states, "The x-ray is at least ten or twelve days behind the actual bone infection."

Great care should also be used in cleaning the wound by removing all infected, soft tissue. Following the advice of Dr. Adson of the Mayo Clinic, I have painted the surface of the dura with 5 per cent iodine. The incision should certainly be wide enough to uncover all bone that might be diseased. The incision that I have used runs from the outer edge of one eyebrow to the outer edge of the other and is bisected by an incision running perpendicular well up into the hairline.

I think one can save a great deal of postoperative convalescence time for the patient by sewing the flaps down in place after the very complete operative removal of all questionable bone. The dura is first painted with iodine and then a gutta percha drain placed over the dura and brought out at the lowest part of the wound. The gutta percha drain can be easily removed, and it is less likely that it will adhere to the dura and thus traumatize it.

REPORT OF CASES

CASE 1.—Boy, aged 13 years, was referred to me by a general surgeon because of a persistent discharge through an opening beneath the left eye near the inner canthus that had been made five weeks previously, because of swelling that extended from the forehead down to the bridge of the nose and over the inner canthus. The incision had been reopened four times in the five weeks, and the mother stated that it had ruptured spontaneously once or twice. He had been swimming nearly every day for one week before the beginning of the swelling; he had had a rather severe frontal sinus pain but was not ill enough to go to bed. The pain subsided when the forehead began to swell. This child had been going to school every day, was not ill and complained of only mild pain when the sinus tract stopped discharging. In fact, the mother said the boy had been doing better work in school that fall than he had ever done before.

Pressure over the swelling on the forehead caused the pus to run out of the incision. I stroked the swelling down and a large amount of pus was discharged. As the swelling receded, my fingers suddenly dropped over the edge of the frontal bone into a soft, mushy feeling area. Needless to say, I discontinued the stroking. The x-ray taken immediately showed a destruction of the frontal bone and also an infected left ethmoid and left antrum and considerable clouding of the right

Fig. 1. Showing rather marked deformity. Flaps were not sewed in place, but were kept separated so wound could be inspected.

frontal. The temperature when admitted to the hospital was 97.8, the pulse was 88, W. B. C. was 7,600, with 3,936,000 reds and 60 per cent hemoglobin and 69 per cent polys. Nasal examination: free pus seen in the nose. Marked swelling of both inferior and middle turbinates.

The following morning, under ether anesthesia, an incision was made running from the outer edge of one eyebrow to the outer edge of the other, with a medium incision extending to the hairline. On elevating these two flaps, a large amount of dura covered with thick granulation tissue was exposed. There were many bony sequestra and the dura was bathed in pus. The bony sequestra were carefully removed and, with a rongeur, the frontal bone was removed sideways and high up. Biting was continued until the bone bled freely. Instead of using a trephine, a small but deep groove was made with a sharp chisel a half inch beyond the rongeured bone. Pus appeared in the groove, and again the rongeur forceps were used to bite away the bone until the groove had been reached, and the rongeuring was continued for another half inch. The bleeding was quite profuse, but was controlled by the use of adrenalin. Both frontal sinuses were found to be involved and the bone was bitten away as far as the lateral extensions of the frontal sinus. The ethmoid cells on the left were filled with pus. The bone was somewhat necrotic in the ethmoid area. The anterior and posterior ethmoid cells were removed through the external incision and the sphenoid was opened widely and was filled with pus. A large intranasal opening was made in the left antrum, which was also infected. The dura was painted with iodine. Iodoform gauze packing was placed over the dura, and the flaps laid down over the iodoform without tying the silkworm sutures that had been previously run through the scalp and periosteum.

The temperature was elevated to 102 following the operation, but gradually subsided to normal at the end of fourteen days. The pulse remained rather rapid during his entire stay at the hospital. He was in the hospital from September 26 to October 19. All the packing was removed the seventeenth postoperative day. It has been a year and a half since the operation, and the boy has had no further trouble. X-rays of the frontal bone show that it is filling in very nicely with new bone.

CASE 2.—A baby, two weeks old, was brought to me because of a protruding left eye and edema of the lids. The extent of the swelling is best seen from the photograph, which will be shown on the screen. The temperature was 103. The swelling was of five days' duration. The cause was unknown. An incision below the left outer external canthus drained about half an ounce of thick, creamy pus. A probe inserted through the opening dropped into the antrum. Uncovered bone could be felt and several small bony sequestra were removed. Irrigation through the small rubber tube placed in this opening caused pus and blood to pour out of the left nostril.

The temperature rapidly subsided. The swelling and proptosis disappeared in a few days' time. The tube was removed at the end of the sixth day. The child made an uneventful recovery. I saw the baby nine months later. There was only a small scar visible below the left external canthus.

CASE 3.—Boy, aged nine years, was brought to me because the mother could feel a soft spot on the top of his head. Examination by palpation showed a round depression in the skull about two cm. in diameter.

The history is as follows:

About a month ago the parents noticed that the child had a general physical decline. His appetite was poor and he ran a mild temperature. About this time he began to complain of a sore spot in the vertex of his head. This became somewhat elevated and he disliked to have anyone touch it. It was elevated to about the size of half a walnut. The elevation gradually subsided and he began to feel better. His temperature was normal, but a week before they brought him to see me the depression at the top of his head could be felt. X-rays revealed a punched out hole, as is shown by the x-rays on the slide. All serologic tests were negative. The boy had very septic tonsils. One month later they were removed. An abscess was found behind the left tonsil containing nearly a dram of thick yellow pus. Culture of pus back of the tonsil: staphylococcus aureus.

X-rays taken at intervals for the next six months showed that the bone was regenerating. This type of osteomyelitis can be classified as xanthic tumors. These generally give the history of being caused by an injury. In this case there was no history of injury, and the bone healed without surgical intervention, but the history on an active nine-year-old boy might be misleading.

CASE 4.—Boy, aged 19 years, with the history that he had been ill in the university infirmary for five days with an acute respiratory infection with temperature, inability to breathe through his nose and marked pain over the left eye. The upper eyelid was badly swollen. The x-rays showed clouding of the left ethmoid and some clouding of the left frontal. His temperature on admission to the hospital was 98.6; pulse 80; W. B. C. was 12,500. Nasal examination showed pus in the middle meatus, considerable swelling of the left turbinate. No

Fig. 2. Taken November 30, 1929, showing large bone defect.

Fig. 3. Taken March 10, 1930, showing beginning regeneration.

Fig. 4. Taken November 11, 1930, showing complete regeneration of skull defect.

Fig. 5. Picture shows much less disfigurement than in Fig. 1. Flaps were fastened down.

pus was seen in the right middle meatus, but there was considerable swelling of the middle turbinate.

Under local anesthetic with intranasal approach, the anterior ethmoids were opened and the frontal sinus was irrigated. Considerable pus was irrigated out of the frontal sinus. The swelling began to subside the next day and the pain was greatly relieved. Four days later swelling began over the right eye, and there was marked pain over the frontal sinus. The temperature was not high, going to only 99. The anterior ethmoids were opened on the right side, and the right frontal was irrigated and thick pus came out of the right frontal sinus. The swelling rapidly subsided and the boy was allowed to go home five days later, having no pain and very little tenderness. Ten days later he returned from his home, a distance of 150 miles, with swelling over the forehead, some bogginess, pain and tenderness over the frontal bone. The temperature was not over 99. The x-ray technician stated that the x-ray showed a beginning osteomyelitis. An incision extending from the outer edge of each eyebrow across the midline, and a second incision perpendicular to this was made from the middle of the forehead to the hairline. There was definite osteomyelitis of the frontal bone. The dura was exposed and was covered with a grayish exudate. A large area of dura was uncovered. The frontal bone was bitten away with a rongeur until free bleeding occurred. The dura was painted with 5 per cent tincture of iodine. A gutta percha drain was placed over the dura and a small iodoform wick was placed over this, both being brought out through the lowest part of the incision. The scalp and periosteum were sewed lightly over the drains; that is, the stitches were not drawn together tightly. There was very little postoperative reaction. The boy made an uneventful recovery and left the hospital on the nineteenth day.

He has had no further trouble. A recent x-ray showed a regeneration of the frontal bone.

SUMMARY

None of these four cases can be classified as the very malignant type of oteomyelitis; however, I feel that the timely and radical intervention in case four stopped what would have been a rapidly developing type of osteomyelitis. The organisms found were pneumococcus in case one, hemolytic streptococcus in case two, and short chain streptococcus in case four.

I think it is safe to close a scalp wound. The convalescence is certainly much shorter and the disfigurement is much less, as is shown by the difference in the photographs of cases one and four. If we are to get further osteomyelitis developing in the bone, keeping the scalp wound wide open will not prevent it, and the early swelling and fluctuation will tell us that we are in trouble as soon as if we were constantly elevating our flap wound to see whether we were having an extension of our osteomyelitic process.

The postoperative x-rays show definitely that we should have no fear about the regeneration of the bone in the cranial vault.

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Symposium
Before the American Otological Society, Detroit, Mich., May 28, 1936

SUBJECT: HEARING BY BONE CONDUCTION

I. Introductory Note.

II. Anatomical, Physiological and Pathological Observations.

(1) Stacy R. Guild, Ph.D. ..... Baltimore
(2) Edmund P. Fowler, M.D., and
Edmund P. Fowler, Jr., M.D ..... New York City
(3) Leroy M. Polvogt, M.D., and
John E. Bordley, M.D. ..... Baltimore
(4) Douglas Macfarlan, M.D. ..... Philadelphia
(5) Hallowell Davis, M.D. ..... Boston

III. Clinical Observations.

(1) George E. Shambaugh, Jr., M.D. ..... Chicago
(2) William J. McNally, M.D., T. B. Erickson, M.D.,
R. Scott-Moncrieff, M.D., and
D. L. Reeves, M.D. ..... Montreal

IV. Report of the Committee on Methods of Testing the Hearing by Bone Conduction.

V. Animal Experiments.

(1) E. G. Wever, Ph.D., and
Charles W. Bray, Ph.D. ..... Princeton University
(2) Heinrich Kobrak, M.D. ..... University of Chicago
(3) E. A. Culler, Ph.D. ..... University of Illinois
(4) Simon Dworkin, M.D., G. A. Hutchison,
M.D., and J. Katzman, M.D. ..... McGill University
(5) Walter Hughson, M.D.,
Eva Thompson, and
E. G. Witting, Ph.D. ..... Philadelphia
(6) Edmund P. Fowler, Jr., M.D., and
T. W. Forbes ..... Columbia University

I. INTRODUCTORY NOTE

We know very little about hearing by bone conduction, but this symposium will be justified if it does nothing more than to arouse interest in the subject and to serve as a basis for future study and discussion.

II. ANATOMICAL, PHYSIOLOGICAL AND PATHOLOGICAL OBSERVATIONS

LII

HEARING BY BONE CONDUCTION: THE PATHWAYS OF TRANSMISSION OF SOUND*

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INTRODUCTION

The question as to which anatomic pathway affords the best transmission of sound waves to the inner ear during tests of hearing by bone conduction has been much discussed in otological literature. The physical structure of the head is such that sound waves, when of sufficient intensity, doubtless do reach the inner ear via all of the pathways, both osseous and osseotympanic, that have been proposed. Therefore, the whole question resolves itself into whether or not transmission via any one of the pathways is better than via the others. For the most part, the arguments on this subject have been based on theoretical considerations alone, and but little objective evidence has been advanced in support of any of the theories.

One result of our ignorance with respect to this phase of the normal physiology of hearing by bone conduction is that the clinical finding of an impairment of hearing by bone conduction is almost universally interpreted as, without question, evidence of a "nerve deafness." The idea that impaired hearing by bone conduction can also be caused by lesions of the best pathway for sound waves to the inner ear has not been given serious consideration in otologic literature.

*From the Otological Research Laboratory and the Surgical Hunterian Laboratory of the Johns Hopkins University Medical School. Aided by a grant from the Ella Sachs Plotz Foundation for the Advancement of Scientific Investigation.

The observations reported in this paper, from the study of human material and from animal experiments, are interpreted as indicating: (1) that for hearing by bone conduction the important pathway to the inner ear is an osseous, not an osseotympanic one; (2) that one of the several osseous pathways that are physically suitable for the transmission of sound waves to the inner ear is of more importance than the others; (3) that this pathway is of more importance than the others because of the direction from which the sound waves via this pathway enter the intralabyrinthine fluids, and (4) that lesions of the important pathway are not infrequent and do cause an impairment of hearing by bone conduction.

REVIEW OF ANATOMY FROM THE STANDPOINT OF PATHWAYS FOR SOUND

Sound, whether heard by air or by bone conduction, is perceived only when the resultant nerve impulses reach the cerebral cortex (chart 1). In the organ of Corti a portion of the physical energy of the sound waves is converted, in some as yet unknown manner, into the physicochemical or biochemical energy of nerve impulses. We know but little about the biologic phenomena concerned with hearing as compared to what the physicist knows about the transmission of sound waves through various media. Nevertheless, the anatomic location of the pathway for nerve impulses from organ of Corti to cerebral cortex is well established, and there is no reason to believe that this part of the pathway is different for hearing by air and by bone conduction.

The pathway to the inner ear that is normally the best for air-borne sounds is shown on the left-hand side of chart 1. Sound waves transmitted along this pathway always enter the intralabyrinthine fluids and approach the organ of Corti from but one direction; this fact is indicated by the single arrow between *Stapes* and *Intralabyrinthine Fluids* and between the latter item and *Organ of Corti.* However, it is entirely possible that air-borne sounds, especially when they are of more than moderate intensity, also reach the inner ear through all pathways that are available for hearing by bone conduction. Physicists have proven that at any surface a part of the energy of sound waves is reflected and the rest is absorbed. The portion that is absorbed is either converted into heat or is transmitted through the new medium as sound. The coefficient of absorption for integument, as for other materials, doubtless is different for different vibration frequencies of sound. So far as I am aware, the values for integument have not been determined. Nevertheless, this possibility, which is indicated in chart 1 by the arrow between *Air* and *Integument*,

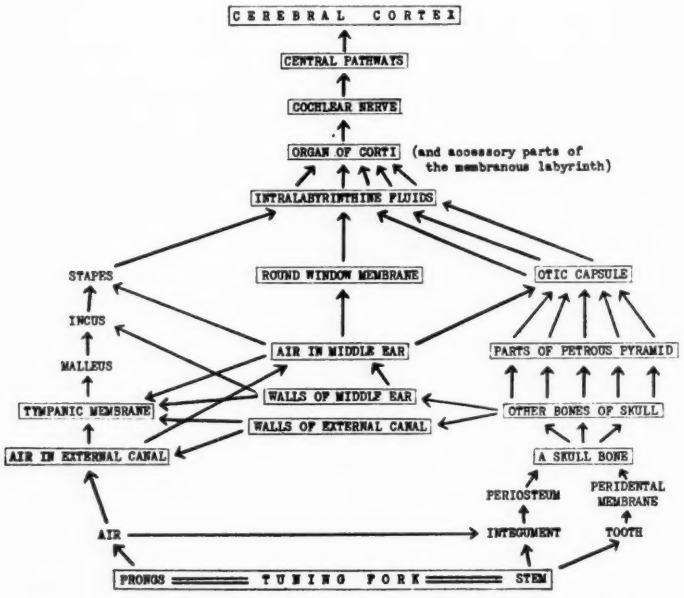

Chart 1. A schematic representation of the numerous anatomical pathways by which sound waves generated by a vibrating object, such as a tuning fork, may be transmitted to the inner ear. There is but one pathway from the organ of Corti to the cerebral cortex.

should be kept in mind, especially when examining patients whose threshold of hearing by air conduction is markedly impaired because of middle-ear lesions or of otosclerosis.

By the strictly osseous pathways to the inner ear, indicated in chart 1 along the right-hand column of items, sound waves may enter the intralabyrinthine fluids from several directions, and thus approach the organ of Corti from several directions. This fact is represented in the chart by the groups of three arrows between *Otic Capsule* and *Intralabyrinthine Fluids* and between *Intralabyrinthine Fluids* and *Organ of Corti.* The total number of directions from which sound waves may reach the organ of Corti is actually much greater than three, as can be demonstrated with a skull and as is indicated in chart 2.

The basic idea that is common to all of the so-called osseotympanic pathways that have been proposed is that the sound waves are transferred from the bones of the skull to the air-conduction mechan-

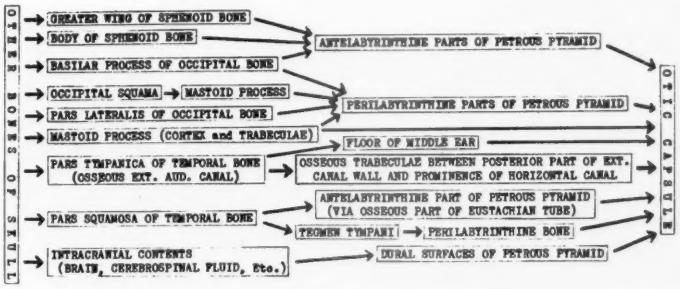
OTHER BONES OF SKULL
GREATER WING OF SPHENOID BONE
BODY OF SPHENOID BONE
BASILAR PROCESS OF OCCIPITAL BONE
OCCIPITAL SQUAMA
MASTOID PROCESS
PARS LATERALIS OF OCCIPITAL BONE
MASTOID PROCESS (CORTEX and TRABECULAE)
PARS TYMPANICA OF TEMPORAL BONE (OSSEOUS EXT. AUD. CANAL)
PARS SQUAMOSA OF TEMPORAL BONE
INTRACRANIAL CONTENTS (BRAIN, CEREBROSPINAL FLUID, Etc.)
ANTELABYRINTHINE PARTS OF PETROUS PYRAMID
PERILABYRINTHINE PARTS OF PETROUS PYRAMID
FLOOR OF MIDDLE EAR
OSSEOUS TRABECULAE BETWEEN POSTERIOR PART OF EXT. CANAL WALL AND PROMINENCE OF HORIZONTAL CANAL
ANTELABYRINTHINE PART OF PETROUS PYRAMID (VIA OSSEOUS PART OF EUSTACHIAN TUBE)
TEGMEN TYMPANI
PERILABYRINTHINE BONE
DURAL SURFACES OF PETROUS PYRAMID
OTIC CAPSULE

Chart 2. An elaboration of that portion of chart 1 between the items *Other Bones of Skull* and *Otic Capsule*. By osseous pathways sound waves are transmitted to the inner ear from several directions.

ism of the middle ear. The several theories of osseotympanic conduction differ from each other only with respect to where and how this transfer of energy occurs. In chart 1 the essential points of difference between the theoretically possible osseotympanic pathways are indicated by the arrows that point toward the left between items of the right-hand, center and left-hand columns. For some of these pathways the theoretical place of transfer of the acoustic energy to the air-conduction mechanism of the middle ear is more specifically indicated in chart 3. By all of the osseotympanic pathways sound is supposed to enter the intralabyrinthine fluids either through the footplate of the stapes or through the round window membrane. The latter structure is, of course, also a possible portal of entry to the inner ear for air-borne sounds.

OBSERVATIONS ON HUMAN MATERIAL*

The collections of clinical records and of temporal bone sections in the Otological Research Laboratory of the Johns Hopkins University have been used in this study. The source of material, method of clinical examination, method of histologic preparation, etc., are adequately described in the paper by Crowe, Guild and Polvogt[1] on lesions responsible for impaired hearing for high tones.

*The studies on human material were presented at the St. Louis meeting of the American Association of Anatomists, April 20, 1935: an abstract of this paper is in the Proceedings of this Association (Anat. Rec., vol. 61, Suppl., p. 20, March, 1935). They were also presented at a staff meeting of the Department of Otolaryngology of the Washington University School of Medicine at St. Louis, April 18, 1935, and were included in a talk before the Section of Otology, New York Academy of Medicine, December 18, 1935.

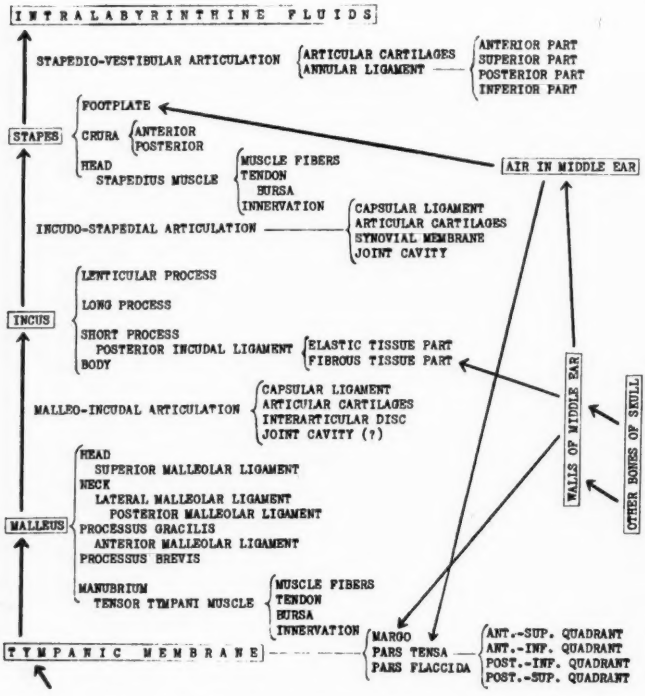

Chart 3. This elaboration of a portion of chart 1 emphasizes the complexity of the anatomical structure of the air-conduction mechanism of the middle ear. Portions of some of the anatomically possible osseotympanic pathways for sound are also shown.

There is but one auditory pathway from organ of Corti to cerebral cortex. Therefore lesions of the inner ear, cochlear nerve or central nervous system should, theoretically, always impair the hearing by air conduction as much as by bone conduction. The histopathologic studies of the temporal-bone sections confirm this view. No lesions have been found, either in any part of the inner ear or of the cochlear nerve, that are peculiar to the ears of patients who had a marked impairment of hearing by bone conduction and but little or no impairment by air conduction.

The structures of the middle ear are part of all osseotympanic pathways. Therefore middle-ear lesions should affect the acuity of hearing by bone conduction in case the best transmission of sound to the inner ear is via any osseotympanic pathway. Theoretically, some lesions might improve and others impair the transmission of sound

Fig. 1. Photomicrograph of a vertical section through the region of subaditus trabeculæ. The rectangular area that is marked out is reproduced at a higher magnification in figure 2. Immediately above the rectangular area are the tip of the short process of the incus and the medial and lateral components of the posterior incudal ligament. The bony wall of the external auditory canal of this ear is pneumatized.

and thus the hearing by bone conduction. From this point of view the material has been studied for evidence of the best pathway. Because of the several theoretical possibilities, the histologic studies included not only the major structures of the air conduction mechanism that are listed in chart 1, but also each of the accessory structures that are listed in chart 3. Without doubt, the hearing by bone conduction remains good in the presence of many middle ear lesions that cause an impairment by air conduction. On the other hand, I have been unable to find any lesions of the air conduction mechanism of the middle ear that are peculiar to the ears of patients who had a marked impairment of hearing by bone conduction and but little or no impairment by air conduction.

With respect to the question of the best pathway for hearing by bone conduction, the above observations, both on the inner ear and on the air conduction mechanism of the middle ear, are essentially negative findings. However, the fact that they are negative adds to the significance of the following observations:

A group of small osseous trabeculæ connect the medial part of the posterior wall of the external auditory canal to the inferolateral aspect of the prominence of the horizontal semicircular canal. These trabeculæ are located in the lower part of the aditus ad antrum, behind and below the tip of the short process of the incus and the posterior incudal ligament (Fig. 1). The normal anatomy of the region is exceedingly variable: the distance to be bridged by the trabeculæ varies from less than 2 mm. to about 5 or 6 mm.; and the size and shape of the individual trabeculæ, the number of trabeculæ and the pattern of the interconnections between them are different in different ears. For the sake of brevity I shall refer to the group as the sub-aditus trabeculæ.

Fractures of one or more of the sub-aditus trabeculæ, with fibrous union of the broken ends, are present in the sections of many ears (Figs. 2, 4 and 7). But rarely are the broken ends of these trabeculæ united by callus. The fractures of individual trabeculæ are sometimes total and sometimes partial. These minute fractures, whether total or partial, are in section more or less similar in appearance to the fractures (Spalten) of the otic capsule that have been reported by Otto Mayer[2] and others, that can be found in any large collection of human temporal bone sections. Although the appearance of the healed fractures, whether of the osseous trabeculæ or of the otic capsule, differs from case to case and from section to section, yet the structural organization of all of them makes it possible easily to differentiate the true fractures from postmortem artefacts. From their histologic appearance it is certain that the fractures that are regarded in this paper as significant were already present at the time the examinations of hearing acuity were made.

The variations in the normal anatomy of the sub-aditus region, together with the fact that many trabeculæ are located obliquely with reference to the plane of sectioning of the temporal bone (Fig. 3), make it necessary to study the serial sections of the entire region in order to determine the extent to which the fractures interrupt the continuity of the sub-aditus trabeculæ as a group. These facts also make it difficult to prove to others, by means of a limited number of photomicrographs, that the lesions in two ears are essentially

Fig. 2. Shows, at a higher magnification, the fractures of the osseous trabeculæ marked off in figure 1. There is a loose but well-organized fibrous tissue between the broken ends of the bone. Study of the serial sections of this ear shows that all of the subaditus trabeculæ have similar fractures. The patient, 47 years of age, had a greatly impaired hearing by bone conduction and but a slight impairment by air conduction.

different, even though similar conditions are present in some of the single sections from each ear.

Mental images of conditions in the whole network of sub-aditus trabeculæ, obtained by careful study of the serial sections, are the basis for the statements in this paper: wax reconstructions have not been made.

Variations in normal anatomy are probably a factor in the frequency of occurrence of fractures of the sub-aditus trabeculæ, but do not seem to be directly related to impaired hearing by bone conduction. Therefore I will not, on this occasion, elaborate the brief description already given of the structural relationships of this region and their normal variations.

The extent of the fractures of the sub-aditus trabeculæ and the acuity of hearing by bone conduction do seem to be related. The facts are as follows:

1. The clinical records show that there was a marked degree of impairment of hearing by bone conduction for those ears in which

the sections show that all of the trabeculæ of the sub-aditus region have complete fractures (Figs. 1, 2, 3, 4). In such ears there was, at the time of the hearing examination, no continuous osseous pathway for sound waves by means of any combination of the trabeculæ between the medial part of the posterior wall of the external auditory canal and the inferolateral aspect of the prominence of the horizontal semicircular canal. The other osseous pathways to the otic capsule are intact in these ears.

2. For those ears that do have a continuous osseous connection across the sub-aditus region by means of even a single trabecula or by some combination of interconnecting trabeculæ, the records show either normal or, at most, a but slightly impaired acuity of hearing by bone conduction, unless the hearing by air conduction was also impaired.

3. For the ears with a conductive type of impairment of hearing by air conduction and a normal, or better than normal, bone conduction time for the 512 d.v. tuning fork, the sections show that the sub-aditus system of trabeculæ is either entirely intact or has a substantial portion that is intact (Figs. 6 and 7).

4. Some people with good hearing bilaterally by both air and bone conduction nevertheless lateralize the sound from midline points to one ear. In many instances the sections of the two ears from such patients show that the lateralization was to the ear in which the system of sub-aditus trabeculæ is either heavier or is more nearly intact than in the opposite ear. Our collection also contains the ears of a patient with unilateral chronic suppurative otitis media who nevertheless definitely lateralized the sound to the healthy ear; the sections show that the sub-aditus trabeculæ are intact in the healthy ear but are fractured in the ear with the otitis media, and that the two cochleæ are equally good histologically. This is, so far as I know, the first time that any lesion has been found that might explain an instance of the so-called "paradoxical Weber test" in a patient with unilateral otitis media.

5. In general, or on the average, the frequency of fractures of the trabeculæ increases with advancing age. The sections of the ears of young adults show that usually the trabecular system of the sub-aditus region is entirely intact (Fig. 5) or has only partial fractures of some of the trabeculæ. I have not seen even a partial fracture of one trabecula in the sections of ears from patients under fourteen years of age, and I have not found a specimen from any patient under forty years of age which has complete fractures of all of the tra-

Fig. 3. This ear, which had normal hearing by air conduction but a marked impairment by bone conduction, has fractures of all of the network of trabeculæ between the medial part of the posterior wall of the external auditory canal and the inferolateral aspect of the prominence of the horizontal semicircular canal. The section reproduced shows fractures of three trabeculæ, only one of which is included for its entire length in this section.

becular connections between the medial end of the posterior wall of the external auditory canal and the prominence of the horizontal semicircular canal.

The preceding observation is in good agreement with one made by Ciocco during his studies of the clinical records of patients examined in this laboratory. Ciocco[3] reports, from an analysis of the hearing tests of 907 patients, that on the average the bone conduction time for the 512 d.v. tuning fork decreases with each decade beyond forty years of age; the patients of each age group had equally good hearing by air conduction for this frequency. In a later study, the report of which is now in press, the same author analyzes the clinical records for 896 ears with impaired hearing by air conduction and with bone conduction time for the 512 d.v. tuning fork greater than air conduction time (Rinne negative); for this group of patients also

Fig. 4. A higher magnification of the area marked out in figure 3.

age is an important factor in the acuity of hearing by bone conduction.

HYPOTHESIS

The above described observations, both the negative findings with respect to the structures of the inner and the middle ear and the positive findings with respect to the trabeculæ of the sub-aditus region, are facts. For the associations that these facts show with the data as to relative acuity of hearing by bone conduction I can advance only an hypothetical explanation. Final proof that this explanation is either correct or incorrect must await the gathering of further clinical and laboratory evidence.

The hypothesis is: That for some unknown reason the organ of Corti is more effectively stimulated by sound waves which approach it from a certain direction through the intralabyrinthine fluids than it is by sound waves from other directions; and that the sound waves which reach the otic capsule by way of the sub-aditus trabeculæ are more effective in stimulating the organ of Corti than are sound waves that reach the otic capsule through other osseous pathways because the sound waves through the sub-aditus trabeculæ are transmitted to the intralabyrinthine fluids from a more favorable direction than are the sound waves via the other osseous pathways to the otic capsule.

The following facts and arguments afford additional support to this hypothesis:

Normal people hear best by air conduction. Sound waves via the air conduction mechanism of the middle ear enter the perilymph through the footplate of the stapes, which is part of the lateral wall of the vestibule and is near the place where the scala vestibuli opens into the antero-inferior part of the vestibule. The organ of Corti is, without doubt, effectively and normally stimulated by sound waves that approach it from this direction.

The sub-aditus trabeculæ are in an almost direct line with the lower part of the posterolateral wall of the vestibule (Fig. 8). From this relationship it seems probable that sound waves conducted via the sub-aditus trabeculæ are transmitted to the perilymph through that part of the vestibular wall between and below the orifices of the two ends of the horizontal semicircular canal. This area of the vestibular wall is near the footplate of the stapes and is almost opposite the opening of the scala vestibuli into the vestibule. Furthermore, the sacculus and the utriculus are so located that there is a continuity of perilymph in the direct line from this region of the wall of the vestibule to the orifice of the scala vestibuli, just as there is from the footplate of the stapes to the scala vestibuli. The combination of anatomic arrangements is such that sound waves transmitted to the otic capsule via the sub-aditus trabeculæ approach the organ of Corti from a direction and in a manner more nearly like sound waves through the air conduction mechanism than do those that come to the otic capsule via the other osseous pathways.

More acoustic energy is required for a normal person to hear by bone conduction than by air conduction. The threshold values, in terms of absolute physical units of energy, are fairly well established for hearing by air conduction, but for bone conduction, so far as I am aware, they have not been determined, even approximately. Therefore the amount of difference is unknown. However, it is easily demonstrable that there is a difference, and that the acoustic energy required for a normal person to hear a sound by air conduction is but a small fraction of the acoustic energy that must be transmitted to the head in order that the sound be heard by bone conduction. One method by which this can be demonstrated is to use either the stem of a tuning fork or a bone conduction receiver as the source of sound for both the air and the bone conduction tests. Another method is for one person with normal hearing to listen through a stethoscope applied to the forehead of a second normal person whose hearing by bone conduction is being tested with an instrument on the mastoid process. Even when the intensity of

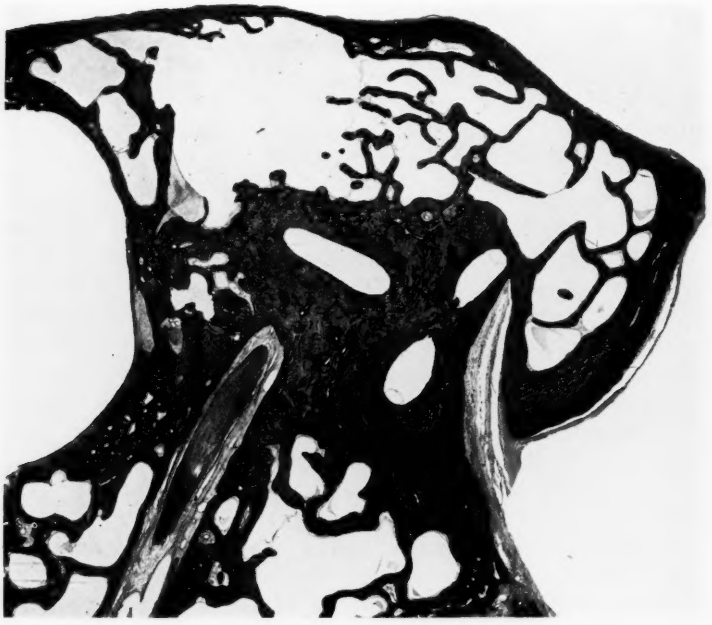

Fig. 5. This ear has, in comparison to the two preceding ones illustrated, relatively thick and short trabeculæ in the subaditus region. There are no fractures. The patient, 18 years of age, had normal hearing by both air and bone conduction.

the test sound is considerably below the threshold of hearing by bone conduction the sound transmitted through the bones of the head can be heard by the person using the stethoscope. There is good reason, from consideration of the physical structure of the skull, to believe that under such circumstances bone conducted sound waves are still reaching the otic capsule of the person being tested, not only from one but from several directions (see chart 2). Although the proportion of the acoustic energy that actually reaches the inner ear has not been established by physical measurements, certainly the deduction is inescapable that even when transmitted by the best pathway for bone conduction more acoustic energy is required for effective stimulation of the organ of Corti than is required for hearing by air conduction.

There is but little doubt that the greater sensitivity of the normal ear to air conducted sounds is in part due to the efficiency of the tympanic membrane and ossicular chain in matching the respective

Fig. 6. This 43-year-old patient, with a chronic otitis media, heard better by bone conduction than by air conduction. By bone conduction the patient heard the tuning fork longer than did the examiner. The uppermost of the three sub-aditus trabeculæ seen in this section is fractured.

acoustic impedances of air and of intralabyrinthine fluids. However, the evidence presented above with respect to the sub-aditus trabeculæ indicates that this is not the entire explanation of the differences in sensitivity of hearing by air and by bone conduction.

The hypothesis advanced above with respect to the physiology of hearing by bone conduction offers, I believe, an explanation that is in accord with all the facts of observation.

Fig. 7. A higher magnification of the area marked out in figure 6.

ANIMAL EXPERIMENTS*

Animal experiments would have no place in this investigation were it possible unequivocally to conclude from the observations on human material that the best pathway is an osseous one. The only osseotympanic pathway that might be impaired by the fractures of the sub-aditus trabeculæ is the theoretical one in which the transfer of energy from the bones of the skull to the ossicular chain is through the posterior incudal ligament. From their close relationships, anatomically (Figs. 1 and 3), it is possible that the trabecular fractures permit a relaxation of the normal tension of the posterior incudal ligament, and thus interfere with the transfer of sound waves to the ossicular chain.

The animal experiments, made with cats, were designed to test the possibility that in these animals the "bone conduction route" for sound waves may be primarily via any osseotympanic pathway, including the one through the posterior incudal ligament. In cats,

*The animal experiments were reported at the Durham meeting of the American Association of Anatomists, April 11, 1936: an abstract is published in the Proceedings of the Association (Anat. Rec., vol. 64, Suppl., p. 18, March, 1936).

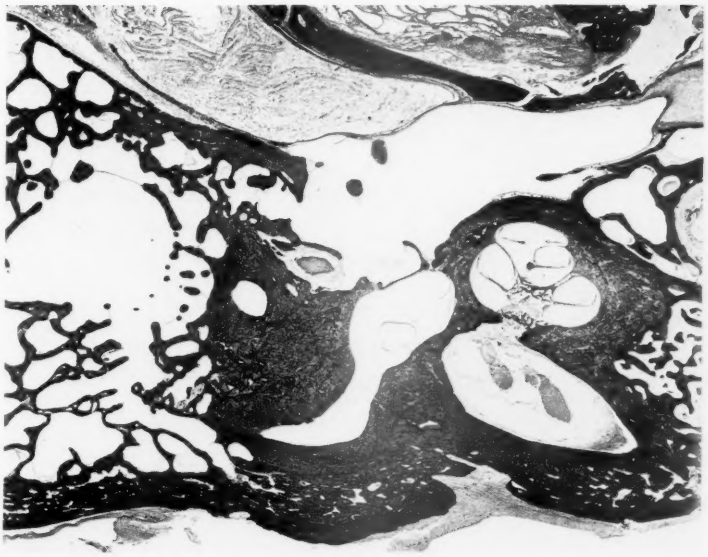

Fig. 8. A horizontal section at the level of the center of the oval window. This plane of sectioning shows the orientation of the subaditus trabeculæ to that part of the vestibular wall near the footplate of the stapes. The orifice of the scala vestibuli is below the plane of this section. (The external auditory canal is filled with impacted epidermal debris.)

as in all mammals except the anthropoid apes and man, the region corresponding to the mastoid antrum and process is not pneumatized: the tympanic bulla is an extension from the hypotympanum, not from the epitympanum. One result of this anatomic difference is that in cats, as in other laboratory animals, trabeculæ corresponding to those of the sub-aditus region of man are not present. In fact, the bone over the arch of the horizontal semicircular canal blends directly into the bone of the outer layers of the skull. Therefore experiments cannot be made that would correspond directly to fractures of the sub-aditus trabeculæ of man.

The Wever-Bray phenomenon was utilized to measure the relative intensities of effective stimulation of the organ of Corti by the sound waves that reached the inner ear before and after interruption of the ossicular chain by an operative removal of the lenticular process of the incus. For the bone conduction tests sound waves from

the stems of magnesium (nonmagnetic) tuning forks and from a hearing-aid type of bone conduction receiver, attached to an audiometer, were transmitted to the heads of the anesthetized cats through a wooden rod twelve inches long. The end of the wooden rod was held firmly against the skin over the lateral end of the occipital ridge, thus simulating, so far as possible, the placing of instruments that is commonly used in clinical tests of hearing by bone conduction. Transmission of sounds through a rod of hard wood twelve inches long does not materially reduce the intensity with which the sound waves are transmitted to the head; this can easily be demonstrated by clinical tests. The rod was used in the animal experiments as a matter of technical convenience when testing with tuning forks: for tests with the bone conduction receiver the length of the rod gives the additional advantage of elimination of magnetic effects from the observations.

The changes in electrical potential produced in the inner ear, as picked up by an electrode in the region of the niche of the round window, were passed through a six-tube amplifier and converted into sound waves by a moving coil type of headphone. Sounds thus transmitted through the normal ear of a cat can be heard by an observer when the source of sound is vibrating at an intensity no greater than is required for threshold perception if the tuning fork or the bone conduction receiver is on the mastoid process of the observer.

The "bone conduction thresholds" for the frequencies of 256 and 512 d.v. were not impaired by removal of the lenticular process of the incus in the ten cats used; but they were slightly impaired, on the average, for the frequencies of 1024 and 2048 d.v. As would be expected, this lesion regularly caused great impairment of the "thresholds" for air conducted sounds of all frequencies.

These experiments indicate that at least for the lower frequencies, the ones most commonly used in clinical examinations of hearing by bone conduction, the transmission of sound waves to the inner ear is primarily via an osseous rather than an osseotympanic pathway.

CONCLUSIONS

Consideration of the combined evidence from the study of the human material and from the animal experiments warrants, I believe, the following conclusions:

1. That for hearing by bone conduction the important pathway by which the sound waves reach the inner ear is an osseous, rather than an osseotympanic, one.

2. That the terminal part of the osseous pathway which is of most importance consists of the osseous trabeculæ that connect the medial part of the posterior wall of the external auditory canal to the inferolateral aspect of the horizontal semicircular canal (termed sub-aditus trabeculæ in this paper).

3. That this osseous pathway is of more importance than are the other osseous pathways to the inner ear because of the direction from which and the place at which the sound waves via the sub-aditus trabeculæ enter the intralabyrinthine fluids.

4. That lesions of this important pathway for the conduction of sound waves to the inner ear cause an impairment of the threshold of hearing by bone conduction.

COMMENTS

While it may be impossible, in the present state of our knowledge, clinically to differentiate with certainty between an impairment of hearing by bone conduction that is due to an inner ear lesion and one that is due to a lesion of the mechanism that conducts the sound waves to the inner ear, nevertheless, it is my opinion, based on the studies reported to you today, that many patients, especially among those over forty years of age, are given a diagnosis of "nerve deafness" when the lesion responsible for this diagnosis actually consists of fractures, with fibrous union, of the osseous trabeculæ of the sub-aditus region. The more correct diagnosis in such cases would be: "Conductive impairment of hearing by bone conduction."

It should be noted that such lesions cannot account for a total loss of hearing by bone conduction any more than lesions of the air conduction mechanism of the middle ear can account for a total loss of hearing by air conduction. It is a common clinical observation that patients with complete absence of the tympanic membrane, the malleus and the incus, and with the stapes covered with dense fibrous tissue, can nevertheless hear by air conduction if the sound is sufficiently intense: doubtless the pathway by which these sound waves reach the inner ear is not the usual one for hearing by air conduction. I believe that an analogous situation exists for hearing by bone conduction, namely, that a lesion of the best pathway impairs the threshold because the only routes then available for the transmission of sound waves are less favorable from the standpoint of effective stimulation of the organ of Corti.

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LIII

NORMAL HEARING BY BONE CONDUCTION AS MEASURED WITH AN AUDIOMETER

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AND
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Bone conduction receivers have been used very little with the electric audiometers in spite of the fact that it was pointed out when the Western Electric audiometer first made its appearance that an audiometric examination is only half completed if bone conduction determinations are not made. We, however, for many years have made bone conduction tests at several frequencies on every patient examined with the audiometer. That is why we were asked to take part in this symposium and to present our data on the "average normal" bone conduction for patients with good hearing by air conduction.

Our report is based on a study of 614 hospitalized patients who had good hearing. The hearing acuity was determined by audiograms made in the regular hospital wards. The patients were often quite ill. From these records we have determined, for each age group (decade), the average threshold for each frequency tested. These data are shown graphically in Fig. 1.

Before discussing the findings it is necessary to state specifically what we mean by "average normal." The zero line of the audiogram chart that is designated as "normal hearing" is, as a matter of fact, the average of the thresholds for eighty-seven healthy adults who were tested in a sound proof room and showed no evidence of ear pathology on otologic examinations. There is no doubt that the zero

*Under grants from the New York League for the Hard of Hearing as well as a grant to the Department of Pathology of Columbia University, from the Hayden-Coakley Fund, for Research in Otology.

line would have been located at a different level on the chart if children had been tested instead of adults, or if the tests had not been made in a sound proof room. Also, it would have been different if more, fewer, or an entirely different group of adults had been used to determine the "average normal."

The heavy black line marked *A* or *BC* on some audiogram charts is the average normal minimum audibility curve for hearing by bone conduction: it was also determined by examinations in a sound proof room. The difference in level between this line and the zero line for air conduction represents the extra power necessary to vibrate the Western Electric D-80904 bone conduction receiver when it is placed against the mastoid processes of the same eighty-seven presumably normal adult individuals.

We have always felt that it was more instructive and much simpler from the standpoint of legibility and comprehensibility if the straight line marked "zero" or "normal hearing" were used as the base line for charting both air conduction and bone conduction. This is very simply done, when using the bone conduction receiver, by subtracting from the actual readings of the audiometer dial the difference in decibels between the lines on the chart representing normal air conduction and normal bone conduction. In other words, the normal line for bone conduction is moved up 35, 45 or 50 decibels as the case may be for each frequency and is thus straightened out and made to coincide with the zero line for normal which is ordinarily used only for AC (See Fig. 2). When both AC and BC are charted from the same point for each frequency one can immediately see the difference between air and bone conduction, and if the bone conduction is not below normal and yet not equal to the air conduction—that is, if we have a mixed type of deafness, it is apparent at first glance also. This cannot be done with tuning forks very well because their decrement changes from second to second, and holding them on the mastoid gives a different decrement than holding them in the air. The audiometer, on the other hand, tests air and bone conduction in the same units and gives quantitatively the actual difference between air and bone conduction.

Therefore in charting our "average normal" bone conduction audiograms it was felt that it would be better to include the air conduction audiograms for comparison. There were many other problems in selection. Since the audiograms were made in the hospital wards they averaged 15 to 20 decibels experimental error (usually below normal), depending on the noise in the ward, the medication

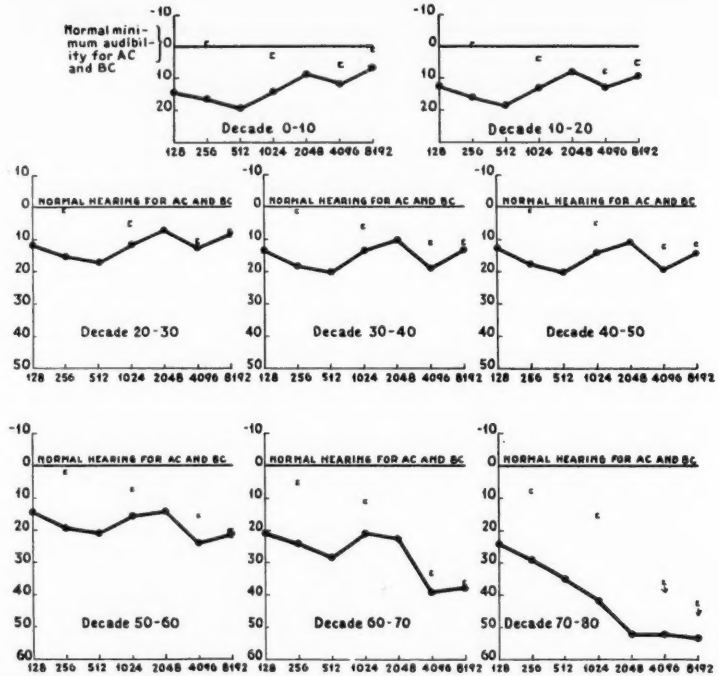

Fig. 1. Average air and bone conduction audiograms for 614 hospital patients who considered their hearing normal. Brackets are bone conduction readings plotted from O line as in normal, heavy black line is the air conduction readings.

of the patient, his ability to pay attention, etc. In all, 1063 audiograms were used in this study. Four hundred forty-nine were discarded either because the difference between the ears was more than fifteen sensation units for air conduction or because the patient had an active otitis or stated that he was hard of hearing or was inconsistent in his responses. All the individuals used in the averages considered their hearing to be normal. Those that had a difference of more than fifteen sensation units for air conduction were discarded because they obviously did not have normal hearing in the poor ear and since no masking was used there is no way of telling whether the bone conduction readings were a record from the more normal ear or across the skull from a poor ear with a conduction lesion. As everyone knows, a conduction lesion is likely to give even better than normal bone conduction readings. The audiograms were made with a 2A Western Electric audiometer by Miss Marie Pless of the New York League for the Hard of Hearing, in the medical and surgical wards of the Presbyterian Hospital and the Babies' Hospital in the city of New York. They were done with the same 2A audiometer which has been loaned to us by the American Society for the Hard of Hearing. No sponge rubber was used about the receiver.

As mentioned before, the audiograms were charted taking an average for each decade and using the ear with the best hearing for air conduction. They show the typical rise at 2048 recorded by Bunch,[2] Ciocco[3] and in another paper by ourselves.[4] They show remarkably good hearing at 256, especially for bone conduction, throughout all decades. The fact that they show good hearing at 256 for air conduction as well argues against the possibility that a large part of the low frequency bone conduction threshold is due to feeling rather than actual hearing. There is some drop, but on the whole surprisingly good hearing, for bone conduction at 1024 throughout all the decades. At 4096 there is a sharp drop for the bone conduction as well as for the air conduction; however, even here BC does not go down as much as AC. The bone conduction drop as the later decades are approached and the pathologic findings at the theoretically proper portions of the cochlea argue for the dependability of the bone conduction apparatus in the detection of nerve deafness. There is also a drop at 8192, although this is not as well marked as the drop for 4096, except in the latest decades. The graphs would show a greater loss at the highest frequencies if the 2A audiometer were more powerful, for in any case where the tone could not be heard at all a value 10 decibels below the loudest sound provided by the audiometer was used in the averages. Undoubtedly some of

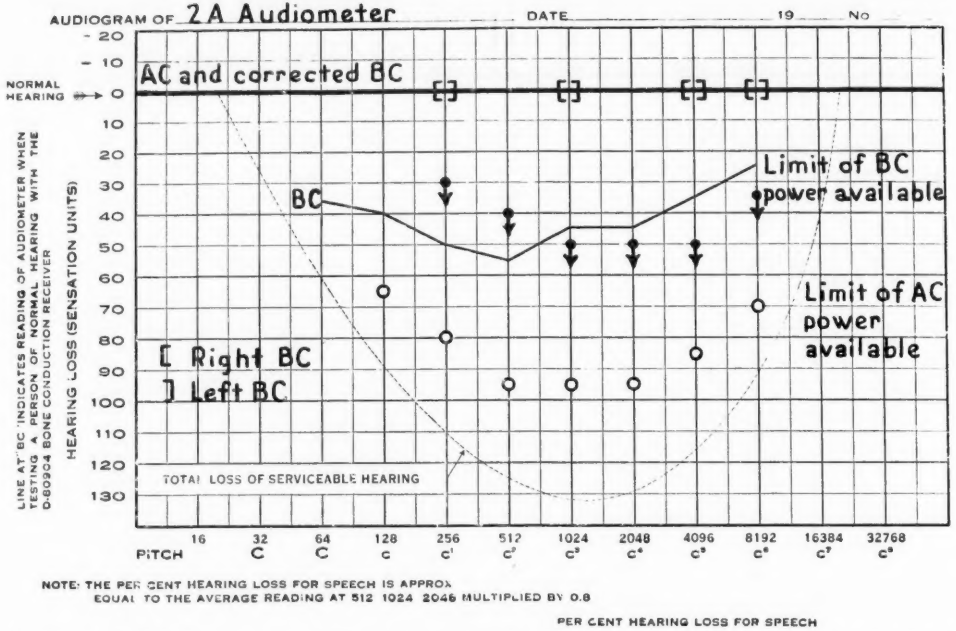

Fig. 2. The limit of A. C. and B. C. power available with a 2A audiometer. Brackets on the normal hearing line show method of recording normal bone conduction for the frequencies used in this study. They are obtained by subtracting 50 for 256, 45 for 1024, 35 for 4096 and 25 for 8192 from the reading obtained on the audiometer dial.

these cases had a much greater loss of hearing than 10 decibels below this level, but we felt unjustified in using a higher figure in our averages.

The marked variation in the individual audiograms emphasizes what we all know, that many individuals can be quite hard of hearing without being aware of it. The patients with good hearing in the sixties and seventies, and even the eighties, make it possible for us to reiterate that age, per se, is not the cause of deafness. We were unable to find much variation in hearing between the sexes, but there was a slightly better hearing for the women than the men. There were small variations between the disease groups. The anemia and blood dyscrasia groups were the best. The arteriosclerosis groups were surprisingly good and the averages for this group, reported in another paper,[4] correspond almost exactly to the entire group here reported. The rheumatic fever group was perhaps the worst of the large groups. Whether this was due to respiratory infection or to salicylate therapy has not been determined.

In the later decades the good bone conduction with poor air conduction for the low notes suggests that, in old age as well as in youth, the commonest and most important deafness is of the conduction type. There is usually a nerve deafness in the higher ranges in old patients, but the greater loss is the conduction loss, and since the lower tones are the speech ranges the conduction loss is the most important one from the standpoint of the patient. This suggests that old people as well as children acquire their deafness from upper respiratory infections; only the old people have had more opportunity to have infections of their middle ears so that more and denser scar tissue has formed at strategic points. It is this scar tissue that makes them hard of hearing and not damage to the cochlea as is generally supposed.

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LIV

PATHOLOGIC CHANGES IN THE MIDDLE EAR OF PATIENTS WITH NORMAL HEARING AND OF PATIENTS WITH A CONDUCTION TYPE OF DEAFNESS*

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AND
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The object of this study is to try to determine the common types and locations of the lesions in the temporal bone that are associated with a conductive type of deafness and a negative Rinne.

First, before attempting to analyze these lesions in ears from patients whose hearing is better by bone than by air conduction, it is necessary to become familiar with the pathologic changes found in the middle ear of patients with normal hearing.

The pathology department of a large hospital provides ideal facilities for the collection of material for an investigation of this kind. With the cooperation of the clinical departments of the hospital, we test the hearing of patients whose general condition makes it seem likely that they will not recover. By this method of selection large numbers of temporal bones are secured from patients whose hearing is normal, as well as from patients with all types of impairment.

Every effort is made to obtain a complete and accurate functional test of the auditory and vestibular apparatus. A detailed otoscopic examination, tuning fork tests, functional test of the vestibular apparatus, and an audiometer test are all made if the condition of the patient permits.

When assembling material for the control group and also for the group having a negative Rinne, a definite standard must be followed. For the control group it is necessary that all tones are heard at an intensity of less than 20 decibels and that the responses to the tuning fork tests are normal. In selecting the group with a negative Rinne we included all ears in which the fork tests, with a 256 or a 512 d.v. steel fork, showed the bone conduction time greater than

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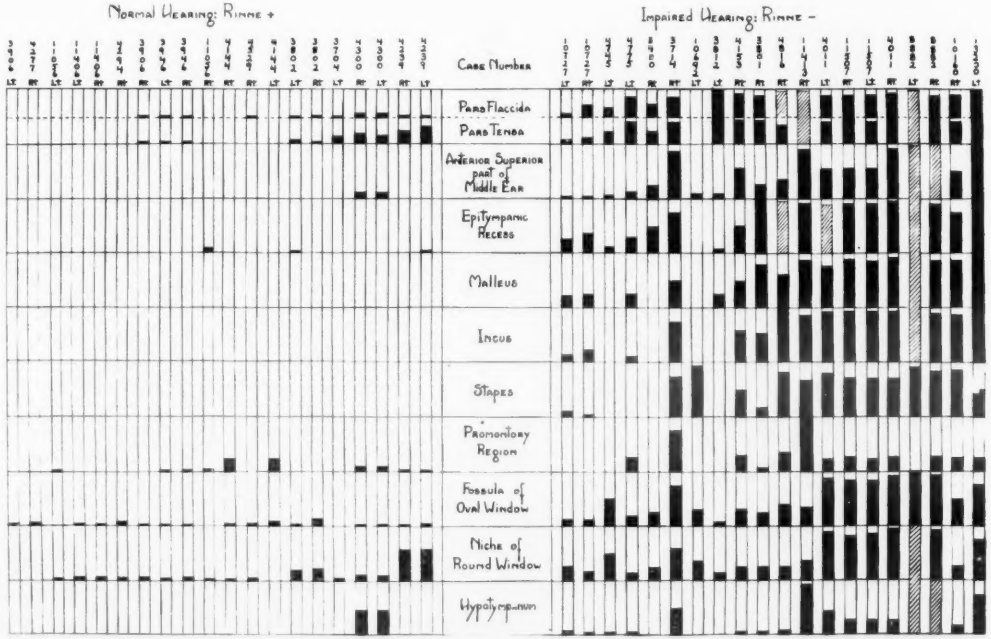

Fig. 1. Chart shows the location and extent of the histologic lesions in twenty middle ears with normal hearing, and in twenty middle ears with impaired hearing and a negative Rinne test. The presence and the extent of the lesions are indicated by the shaded areas.

the air conduction. The bone conduction time of the patients when tested was compared with that of the examiners, whose bone conduction times are normal. These comparisons showed, with the exception of one case, a normal bone conduction time for patients with a negative Rinne. The one exception was a patient sixty-three years of age, and his bone conduction time was only a few seconds shorter than the examiner, who was thirty years old. We included in this group two border line ears in which air conduction and bone conduction were equal.

In the selection of both these groups it is necessary that the time interval between the hearing test and the removal of the temporal bones be as short as possible. It is also necessary for both groups that the preservation of the temporal bones be good enough to permit a positive statement as to whether the structures of the inner ear as well as the middle ear are normal or pathologic.

We studied the records of some 950 tested ears; of this number only sixty-three ears met the requirements for the normal group and twenty ears met the requirements for the group in which bone conduction time was better than air.

The time between the clinical test and death was short in the majority of cases, of both the normal hearing group and those with a negative Rinne. This time interval was less than thirty days for half of the ears with normal hearing, and for the other half averaged three months. For sixteen of the twenty ears that had a negative Rinne the tests were made within six weeks of the date of death; for two the time interval was three months and for two eleven months.

The temporal bones of each of the above groups were sectioned serially and the middle and inner ear structures were studied in detail. Slight lesions in the inner ear of each group were occasionally noted. These changes consisted of a partial atrophy of the nerve, the organ of Corti and the cells of the external sulcus in the last one or two millimeters of the basal coil of the cochlea. These changes we know from previous study to be within normal limits.[1]

For the convenience of study, the middle ear was divided into ten areas. The corresponding areas in the two groups (those with normal hearing and those with impaired hearing) were compared. In judging the amount of histologic change in the various areas special attention was given to the amount of scar tissue, its density, and the extent of bone destruction. The principal types of lesions found in sixteen ears are those characteristic of a long standing chronic

Fig. 2. Thickening and calcification of the tympanic membrane that did not interfere with the hearing of any tone from 64 to 8192 d.v. (Published in the Arch. of Otolaryng., 23:48-56, January, 1936.)

Fig. 3. Extreme thickening of the tympanic membrane due to chronic otitis externa (case 3812 in Fig. 1). This patient had impaired hearing and a negative Rinne test.

infection, with the resultant formation of scar tissue. A cholesteatoma was present in three of these ears. Two ears show evidence of a long standing tuberculous infection. One ear has typical otosclerotic lesions around the footplate of the stapes; another has the entire middle ear filled with a new growth which extends through the petrous pyramid from an intracranial tumor.

Chart 1 is a graphic record of the conditions found in the specified areas of the middle ear. On one side of this chart is illustrated the estimated amount of pathologic changes in twenty of our sixty-three normal ears,[2] while on the opposite side we have shown the pathologic changes in twenty ears with bone conduction time greater than air conduction. The ears with impaired hearing and negative Rinne are arranged according to the degree of their impairment, those with the greatest hearing loss being on the extreme right of the chart.

The graphic representation of these lesions shows in striking fashion that as the hearing by air conduction decreases the pathologic changes in the middle ear increase. It is likewise interesting to note that the only ears in which air conduction equalled bone conduction are the two (10727 left and 4745 left) that are closest to the normal side of the chart, both from the point of view of air conduction acuity and pathologic changes (Fig. 1).

The lesions in the pars flaccida and pars tensa of the tympanic membrane are charted separately and it is interesting that the frequency with which lesions are found in these two anatomic areas are about equal. In the normal hearing group the extent of the pathologic changes varied from slight thickening to marked calcification and from moderate to extreme retraction (Fig. 2).

We find pathologic changes of the drum in nineteen of the twenty ears with a negative Rinne. In this group the lesions are more extensive. The lesion of the tympanic membrane, as illustrated by case 3812 (Fig. 3), was evidently sufficient to cause a negative Rinne, since it is the only marked pathologic change in the middle or inner ear of this case.

The anterior-superior part of the middle ear has pathologic changes in every case in the negative Rinne group. The changes in this area vary from slight thickening of the mucous membrane to the filling of the entire space with fibrous or cystic tissue and often with new bone. Only two of the normal hearing group have any deviation from normal in this region.

Fig. 4. Section through the middle ear of case 11507 Right. There is almost complete destruction of the drum. The ossicles are destroyed except for a small part of the head of the malleus and foot-plate of the stapes. The middle ear is lined with squamous cell epithelium. The epitympanic recess, fossula of oval window and niche of the round window are filled with dense cystic tissue.

Fig. 5. Section through the middle ear of case 10727 Left. The drum is retracted. Adhesions are dense between the head of the malleus and the walls of the middle ear. The space between the malleus and incus is filled with fibrous tissue.

The epitympanic recess of the negative Rinne group shows extensive lesions of the same character as those found in the anterior-superior part of the middle ear. Nineteen of the twenty ears have this area involved (Fig. 4). Seldom is there any change in this area in cases with normal hearing; in fact, only three out of our group show a very slight change from the normal.

The areas in the chart with the most striking contrast between the two groups are those allotted to the individual ossicles. Pathologic changes involving the ossicles are not seen in the normal hearing group. Eighteen out of the twenty in the negative Rinne group show lesions involving one or more ossicles. These changes may be adhesions between two or more of the ossicles, or adhesions between the ossicles and walls of the middle ear (Fig. 5). Erosion of the ossicles, as frequently occurs in chronic infection, is common (Fig. 4). Bony changes in the capsule, due to otosclerosis and ankylosis of the footplate of the stapes, are illustrated by case 10692 (Fig. 6).

A perfectly clear fossula of the oval window and niche of the round window is rarely found in any middle ear. Occasional bands of mucous membrane are found across both these regions in all the normal hearing group. In the negative Rinne group this area is also involved. Here the changes are more extensive, and frequently the entire niche of the round window or fossula of the oval window is filled with fibrous tissue (Fig. 7).

Pathologic changes in the mucous membrane of the hypotympanum are not as striking as in the other areas of the middle ear. In only two ears of the normal hearing group is there any change. Slight changes are seen in fifteen ears that had a negative Rinne, but only two ears in this group had extensive changes.

SUMMARY

1. The histologic lesions in the middle and inner ears of patients with normal hearing and those with a conductive type of deafness and a negative Rinne are compared. The most striking difference is the absence of any lesion of the ossicles in the group with normal hearing, and the presence of one or more lesions involving the ossicular chain in every ear in the group with impaired hearing.

2. We believe that when the Rinne test is negative it is the result of one or more lesions which interfere with the normal movements of the ossicular chain. This causes a reduction of the air conduction time without changing the bone conduction time.

Fig. 6. Section through the footplate of the stapes in case 10692 Left. Otosclerosis involves the footplate and the bone along part of the facial canal.

Fig. 7. Fossula of oval window is filled with very fibrous and cystic tissue. The niche of the round window is filled with the same type of tissue which is adherent to the secondary tympanic membrane.

3. The specimens on which these studies are based did not include the entire eustachian tube, so we have no information about tubal lesions in patients with normal or impaired hearing.

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LV

SKULL RESONANCE

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The story of bone conduction might well be put down as an otologic saga. It seems to emerge from a remote past, the tale being told in a piecemeal, disjointed fashion by one after another of the otologic leaders of former generations. The various contributions do not fit together, nor do they make a free-flowing story which builds up a definite picture. We have at present a jumble of ill-fitting chapters for which the most that may be said is that each is concerned with setting down some fragmentary facts.

All of this leaves us in a puzzled state of mind and causes questions to arise which are more easily put than answered. We have, then, a reasonable excuse in attempting to clarify the matter, resolving some of the simpler of the problems, discarding some of the useless, and setting a course toward more definite objectives.

When making a bone conduction test there is not only a direct transmission of the sound to the end organ, but there is also set up a skull resonance which may greatly alter the test findings. I believe that but few otologists realize that the latter exists. It should be constantly kept in mind that resonance is taking place, no matter where we place the fork (or other sounding body). This resonance is influenced by:

1. Asymmetry of the skull (present in nearly all cases).
2. Density of the bone.
3. Condition of the sinuses.

In addition to these factors that influence resonance we must not forget that we are making subjective tests, asking for and depending upon the patient's responses. With all these factors present, perplexities arise which have been puzzling the leaders in otology for generations. The mere listing of them will give a clue to the confusion of the problem, and will strongly suggest that many pitfalls still exist for one who would make dogmatic otologic diagnoses.

1. The classical test of placing the fork on the vertex usually shows that in catarrhal deafness the patient lateralizes to the affected side, in nerve deafness to the sound side, and in normal hearing individuals there is no lateralization. Yet exceptions are frequently found.

2. Bone conduction tests from the mastoid should follow the same rules; yet many patients with an acute mastoiditis have prolonged B.C. when the fork is held on the mastoid, but lateralize the same fork from midline points to the healthy side. This is the so-called Weber-Schwabach paradox of Mackenzie. Can it be explained?

3. Lateralization from the midline often varies with the testing pitch used (chart 1).

4. In bilateral otitis the value of lateralization is distinctly doubtful (chart 2).

5. If the external canal is stopped up with the finger or if the canal is filled with water, the B.C. time is increased; but if pressure is applied to the column of air or to the water in the canal, B.C. is decreased. These facts bothered Wheatstone in 1827, Politzer in 1864, and later Lucae and Kretschman.

6. A confusion that may arise in testing pure skull resonance is the undoubted transmission of air borne sound into the stopped-up ear by way of the auricle and canal cartilages. This was observed by Helmholtz and Kisch.

7. Many of the older men of Politzer's time were puzzled by finding a small amount of bone conduction on the side on which the labyrinth was known to be destroyed. Little masking was done in Europe then, and the possibility of hearing across the skull was conveniently called "borrowed bone conduction."

These are but a few of the perplexities that confront us daily as we study our cases. They make us conscious of the fact that we

should more often keep an open diagnosis on progressive deafness, otosclerosis, luetic deafness and mixed deafness, as made from test findings alone.

With this preliminary I would ask the consideration of some of the facts of skull resonance in general.

1. By the method in vogue "we depend exclusively on what information we may get from the patient, without being able in the slightest degree to control or to contradict his or her statements." (Nussbaum.)

2. Direct skull phonendoscopy may be described as listening to the resonance of a skull.

(a) One technic used is to place the vibrating body at various locations on the skull and to listen to the resonance through binaural auscultation tubes placed in the patient's and in the observer's ears. (This is a convenient method to detect lateralization in children.) (Chart 3.)

(b) Another method reverses the process and places the vibrating body at an indifferent point (between the incisors or on the vertex or occiput). The observer uses a small stethoscope to listen at different locations.

(c) An absolutely impersonal method is to pick up the resonance and measure its intensity electrically.

The first two methods are practical for any experimentally inclined otologist; they demand, however, an oscillator with a frequency range and a decibel meter. Although fair work may be done by carefully struck and timed forks, instruments capable of maintaining pitch are much to be preferred. With them, the end point of hearing, or the fade-out reported as decibels of attenuation is the index of the amount of resonance at a given location.

If the audiometer bone conduction receiver is used, it must be borne in mind that varying the pressure causes variations of the intensity of the sound. Exact pressure control is never possible, so varying the pressure to seek an optimum response is the solution. Where dried skulls are used, a vibrating pin is screwed into the midline at the occiput, and both the skull and the vibrator are fixed immovably.

These various methods have all been used in obtaining the records herewith reported.

The Contralateral Effect.—In most normal hearing individuals, if a vibrating object is placed upon one parietal eminence, the sound is best heard in the opposite ear; though there are exceptions. Politzer, Lucae, Bezold and Frey all have reported the phenomenon, and the latter had a questionable explanation in the reunion of lines of resonance. We have confirmed the finding that removal of large pieces of the vault does not alter the effect, whereas masses of wax, plasticene or lead attached to the vault do change the lateralization. Intracranial tumors are said to act similarly; but I have never found confirmatory evidence of this. We believe that the determinant factors are those of an enforced resonance and not bone conduction.

The transmission of sound by the skull, as compared to resonance, is undoubtedly less influenced by differences in density, continuity and architectural struts of the bone. Even extreme pathologic changes in the bone, such as occur in hydrocephalus and in osteitis, may affect the transmission, but to a relatively small degree. We are dealing with a sound conductivity greatly more efficient than that of air, and invariably we have an end organ located in a dense bony structure. It might be inferred, therefore, that as far as the patient's reportings are concerned, hearing capacities are recorded. But resonance is one factor that greatly influences these reportings. For example, the condition of the larger nasal sinuses frequently determines the lateralization of sound. This fact is often overlooked, and yet it is easily observed. The condition of the larger nasal sinuses not only influences lateralization but also the bone-conduction threshold for forks and audiometer (Austin Hayden); so that, just as masking must be taken into consideration in testing, so perhaps should resonance when we are evaluating B.C. findings.

The principles of resonance should be considered in this matter. The initial effect of a resonator is to reinforce sound, but it also uses up acoustic energy, as may be demonstrated with the forks. For this reason Fowler recommends that the "on and off" method be used in testing B.C. with tuning forks. A 488 d.v. fork, struck a standard blow, is heard normally for 55 seconds. When placed immediately and continuously on a resonation box the hearing time is reduced to 40 seconds, and when placed "on and off" the box at three-second intervals, the reduction from normal is only three seconds.

With the fork on the patient's vertex and binaural tubes in patient's and examiner's ears the following observations are obtained:

(a) Normal cases report hearing as long as that of the examiner, and equal in both ears. (It is obvious that the examiner's hearing should be normal.)

(b) Asymmetry of the skull or unilateral empyema of the frontal or maxillary sinus will throw the resonance to the same side for both examiner and examined. Unfortunately, it is not invariably true that a diseased sinus will cause lateralization.

(c) Impacted cerumen or canal occlusion will cause the patient to refer to the affected side, but the examiner will hear less well from this side. This fact supports the argument that there is actual dispersion of sound out of an ear when testing by B.C. The masking effect of extraneous noise scarcely enters into this experiment, as the stethoscope tip is already excluding the room noise.

(d) Unilateral mastoid disease usually gives to both patient and examiner a reference to the diseased side, but this is not an invariable findings. The Weber-Schwabach paradox exhibits a patient's reference to the sound side, in spite of lengthened bone conduction on the affected side. No explanation is offered.

(e) In bilateral lesions of ears or sinuses no dependable or significant findings may be had. (The lesion may be recent on one side and old on the other.)

(f) Uncomplicated unilateral nerve deafness does not alter the resonance to the examiner; it should be equal in both ears, but to the examined the hearing is naturally referred to the good ear.

(g) Uncomplicated unilateral catarrhal deafness should give a reference by both patient and examiner to the diseased ear, although the duration of hearing may be longer for the patient.

This method is very interesting to use, particularly when timing is done, and I believe that skull phonendoscopy is an adjunct to our present methods of testing.

Though it will not be considered here, it is possible for the examiner to check the patient's A.C. by placing the fork on his own head.

The contradictory feature of the method is that with the fork on the patient's head he is reporting his bone conduction, while with the binaural tubes in the ears the examiner is recording his own AC. The odd part of it all is the fairly identical timing for both examiner and examined in normal and catarrhal cases.

Objective examination of skull resonance by auscultation at various regions with the sounding body (a flat bone conduction receiver) held between the teeth, gave the results shown in the following table:

FADE-OUT IN S.U. TO DIMINISHING SKULL RESONANCE WHEN AUSCULTATED AT VARIOUS PLACES

Source of Sound, the Vibrations From a 3-A Audiometer Activating a Gernsbach B.C. Receiver, Held Between the Teeth

		M. W.	W. B.	K. P. R.	J. A.	W. W.	H. L.
Parietal eminences	R.	70-75	80	80	85	90-95	85
	L.	70	80	80	80	90-95	85
Frontals	R.	70	70	80	75	95	90
	L.	70	70	80	80	95	85
Antra	R.	60-65	65	70	65	85	70
	L.	60-65	65	70	75	85	70
Mastoids	R.	75	75	80	80	85	
	L.	75	75	80	75-80	85-90	80

The best resonance appears in auscultating over the maxillary antra; the other locations run roughly equal.

Subjective reporting upon B.C. fork hearing from different locations are shown as follows:

B.C. FORK HEARING FROM VARIOUS SKULL LOCATIONS (PITCH, C512; STANDARD BLOW)

Average of Ten Tests on Ten Normal Individuals, the Figures Representing Hearing Time in Seconds, as Reported by Patients

Fork hearing duration from vertex	20 seconds
Fork hearing duration from mastoid antrum	30 seconds
Fork hearing duration from upper teeth	45 seconds
Fork hearing duration with handle inserted lightly in E. A. M. (Mixed A. C. and B. C.)	60 seconds
Fork hearing by A. C. at E. A. M.	75 seconds

In order to obtain absolutely objective findings, Dr. Batson and the author have checked the skull resonance of the dried skull. The skull was activated by an oscillator in contact with a rod that was screwed into the occipital protuberance. Auscultations were made at various points on the calvarium with a Boles stethoscope, to the center of the diaphragm of which had been cemented a thumbtack. The frequency of 512 d.v. was used throughout. Attenuations

CHART 1

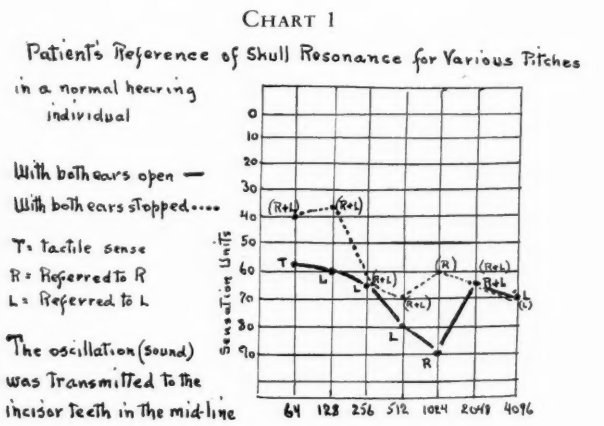
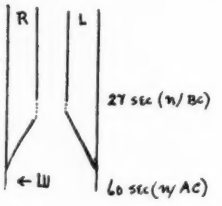


CHART 2

A.R., age 26, bilateral O.M.P., subacute, uncomplicated, tubal catarrh



RBC	35:27
LBC	30:27
RAC	48:60
LAC	48:60

Contradiction in Auscultation

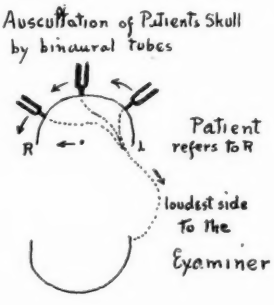
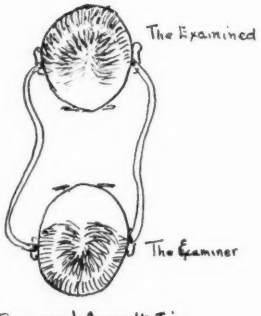


CHART 3

Contralateral Reference of Skull Resonance



Binaural Auscultation

of sound were measured in decibels. The amount of attenuation necessary to produce a fade-out was determined.

It was found that the vertex was the weakest point of resonance, and that resonance increased as the radial lines were followed toward the more dense basal parts of the skull. The contrasts were so marked as to make one suspect that the heaviness and denseness of the bone of the base gave increased transmission. Masses of plasticene or plaster, attached to the inside of the vault, definitely increased the resonance at these points. The inference is that intracranial tumors attached to the bone would do likewise.

In conclusion, it seems reasonable to keep in mind that vagaries in skull resonance may be responsible for subjective lateralization, which may be interpreted by the examiner as individual and uncomplicated responses of the end organ, when this is not wholly the case.

Skull auscultation similarly shows such vagaries and contradictions as to be undependable for absolute findings. Reports of auscultation are, however, of frequent value as confirmatory evidence.

1805 CHESTNUT ST.

LVI

BONE CONDUCTION, VIBRATION, AND ELECTRICAL STIMULATION*

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The fundamental problem in the evaluation of bone conduction is to determine clearly and precisely its relation to the usual mode of excitation of the cochlea by air conduction. It is perfectly evident that the first stages in hearing are physical, namely, the transmission of sound waves from the external medium—whether air or the handle of a vibrating tuning fork held in contact with the head—to the inner ear. It is equally clear that the final stages are nervous and depend upon nerve impulses passing over certain pathways and to certain centers of the brain.

Is it possible that bone conduction depends upon the sense of vibration rather than hearing? In spite of the close relationship of

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the sense of vibration to hearing, there should be no real difficulty in distinguishing between them to the extent of determining to which sense modality and to which centers in the brain the perception of sound by bone conduction belongs. If we determine the range of frequencies which can be detected by ordinary hearing and compare it with the range to which the finger tips are sensitive, we find that, although there is considerable overlap, nevertheless we can hear high frequencies that cannot be felt and can feel vibrations that are too slow to be heard. If we go further and determine what change of frequency or of intensity we must make in order that it be detected by finger and by ear, we find that, although the finger is nearly, if not quite, as acute as the ear in detecting differences of intensity, it is extremely crude by comparison in the recognition of differences in frequency.[8]

By this test of differential sensitivity, it is clear that bone conduction, as we use it in tests of audition and in devices to aid hearing, is actually an approach to the sense of hearing, and not to the sense of vibration. A more direct, if also more naive proof of the same proposition is that we can immediately interpret speech or recognize a tune through a bone conduction receiver. They "sound" essentially the same. We cannot do this by touch, even though the work of Gault, and that of Goodfellow,[7] in training deaf children to recognize sounds through their finger tips shows that a deaf person may, to some extent, learn to use the sense of touch to replace a lost sense of hearing, precisely as a blinded man may learn to read by touch the raised letters which he formerly read by sight.

Although many weird theories have appeared in the field of audition, I doubt whether anyone has ever supposed that bone conduction might excite the hearing centers of the brain directly by mechanical agitation. A possibility only slightly less remote has been seriously considered, and that is that all hearing by bone conduction is due to direct mechanical stimulation of the auditory nerve at some point beyond the organ of Corti. And within the realm of plausibility is the chance that, although normally bone conduction is simply the transmission of sound by an unusual route to the organ of Corti, which then excites the auditory nerve in the usual way, nevertheless, if the organ of Corti be damaged, more intense vibration transmitted by bone conduction might be heard through direct mechanical excitation of the nerve. This is closely related to the possibility of direct electrical stimulation of the auditory nerve.

As long as it was possible to believe that the pitch of a tone was recognized because it set up in the auditory nerve impulses of the

same frequency as the incident sound waves, it was possible to hope that an electrical or mechanical ear might be invented to replace a damaged cochlea and set up the desired frequencies of nerve impulses by direct stimulation. The discovery by Wever and Bray that the frequencies of the stimulating sound could actually be recovered electrically from the auditory nerve gave strong support to this so-called "telephone theory." As experimental analysis proceeded, however, it became evident that it was most improbable that the theory could apply to high tones. At one stage of our own investigations I was strongly impressed with the telephone theory as applied to tones below 800 c.p.s., but now I believe that it is completely untenable, even in this restricted form. The evidence and arguments have been published elsewhere, and most of them have been presented before this Society,[2 10] and need not be reviewed in detail. I merely remind you of the positive evidence presented at last year's symposium in favor of the alternative theory of resonance and localization on the basilar membrane, and shall call to your attention some observations showing the independence of subjective pitch from the physical frequency of the sound waves.

The fundamental fact is that if we hold the frequency of a tone constant and vary its intensity it undergoes at the same time, subjectively, a change of pitch. Many scattered observations by numerous investigators were confirmed and elaborated by my colleague, Dr. S. S. Stevens,[9] in 1935, and again almost immediately thereafter by Fletcher[3] and his collaborators in the Bell Laboratories. The effect is greatest for very high and for very low tones. High tones seem to become higher and low ones lower as they are made louder. The effect is slight and more complicated for intermediate tones. It is particularly prominent for low tones, which are precisely the ones for which, were the telephone theory true, it would be impossible. This final remnant of the telephone theory, and with it the possibility of a mechanical or electrical substitute for the cochlea, must therefore be abandoned.

We must, nevertheless, explain the fact which has been independently reported by several observers, and which we have ourselves confirmed, that an alternating current introduced into the ear may produce the sensation of a musical tone of a pitch corresponding to the frequency of the current employed. To carry out this experiment, the external auditory canal is filled with physiologic salt solution and one electrode is immersed therein, and the circuit is completed through an indifferent electrode on the arm. Some of the earlier experiments were explained as due to mechanical vibration

set up in one way or another at the electrode in the ear, thereby generating audible sound. The experiments of Fromm, Nylén and Zotterman[4] and of Gersuni and Volokhov[5] apparently eliminated this possibility. The Russian investigators present good evidence that a single shock applied in this way does actually stimulate the auditory nerve directly.[6] It is heard by the subject as a sharp noise or click. We might therefore suppose that the alternating current likewise stimulates directly, and that the cochlea is not the necessary organ of hearing that we have argued it must be. Fortunately for our peace of mind and also for our theory, Andreef, Gersuni and Volokhov[1] performed the final critical experiment of stimulating electrically ears whose cochleas had been destroyed by disease or accident. In this case a single shock still gives a sharp noise, but alternating currents are not heard as musical tones. They too become merely noise. This is precisely what the place-resonance theory demands on the assumption that the electric current stimulates at random fibers corresponding to various parts of the basilar membrane. When the cochlea is present we can best explain the hearing of the alternating current as a musical tone as due to mechanical agitation induced in the inner ear in some unknown way, which is then analyzed by the basilar membrane and organ of Corti exactly as if it had been induced in the usual way by air conduction.

This problem of electrical stimulation is therefore exactly analogous to bone conduction. Before hearing can occur, the auditory nerve must be stimulated. Before the sensation of pitch or the recognition of a musical tone can occur, a particular group of nerve fibers must be stimulated, and this selection can be made only by the cochlea, because the selective resonance of the basilar membrane and other vibrating structures provide the only mechanical mechanism capable of making the selection. How an alternating electric current or how mechanical vibration of the temporal bone can induce this selective mechanical vibration within the cochlea are both physical problems to be solved in physical terms. Air conduction, bone conduction and electrical conduction may therefore be considered as alternative physical pathways leading to the cochlea with its organ of Corti. Here is an essential organ. There is no substitute for it if the pitches of sounds are to be recognized. Impairment of hearing by bone conduction implies either a lesion of the organ of Corti or of some essential nervous mechanism beyond it, or else failure of the tissues of the head to transmit the physical vibrations. We must admit the possibility of a "transmission deafness" in bone conduction, but so long as a patient recognizes pitches by either air or bone conduction

there must be an organ of Corti present; and whenever there is this clinical proof that the essential end organ is present it is possible that the patient's hearing may be benefited by some form of artificial hearing aid.

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III. CLINICAL OBSERVATIONS

LVII

THE INTERPRETATION OF BONE CONDUCTION AND OTHER TESTS OF HEARING

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CHICAGO

The advent of the audiometer as a convenient and graphic method of testing and recording the hearing has stimulated a renewed interest in hearing tests. Now that we have this new, accurate and standardized method for testing the hearing for the entire tone range, are bone conduction tests still so important in the diagnosis of deafness? If they are important, does not the bone conduction curve, sup-

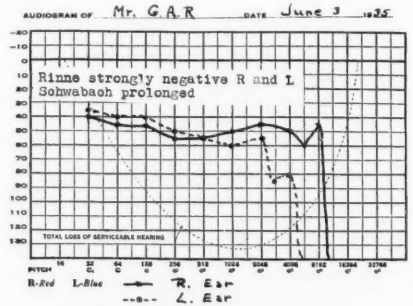
plementing the standard curve of the audiometer, answer all our needs and supplant the old tuning fork tests?

Chart 1.—Here are two similar audiograms. A horizontal type of curve, especially with a depression in the middle tone range, is usually associated with conduction deafness. This first patient has conduction deafness due to otosclerosis. The second patient with an almost identical audiogram has primary nerve deafness.

Chart 2.—Again we have two similar audiograms. This type of curve, with progressive loss for the higher tones, is usually associated with nerve deafness. The first patient has primary nerve deafness. The second patient has a strongly negative Rinne and prolonged Schwabach. The atypical curve is due to an unusual degree of secondary nerve degeneration, yet the fact remains that he has primarily a conduction deafness and is helped by an electrical hearing device, and these facts could not have been diagnosed from the standard audiogram; in other words, the standard audiogram alone cannot be used for the diagnosis of the type of deafness. Bone conduction tests are essential.

Chart 3 (Mrs. L. H.).—This is the standard audiogram supplemented by the bone conduction curve obtained with the Western Electric bone conduction receiver. This patient has unilateral deafness. Notice that in the deafened ear bone conduction is better than air conduction for the low tones; in other words, from 32 to 128 vibrations the Rinne is negative. Now notice the bone conduction curve for high tones, how it seems to rise rapidly in the normal ear until for the very high tones it equals the hearing by air. This simply means that the bone conduction receiver is heard increasingly by air for these high tones. For the upper half of the tone scale the bone conduction curve is, therefore, unreliable and is not really a curve of bone conduction at all. Notice that this patient was unable to hear either the 32 or 64 forks in the hard-of-hearing ear, yet the standard audiogram curve shows apparent hearing for these tones. This is due to an element of bone conduction from the standard receivers which fit snugly against the ears. Thus the standard audiogram, although it is supposed to represent hearing by air conduction alone, actually represents an element of bone conduction sufficient to give a false picture of the hearing for low tones in conduction deafness.

Chart 4.—Here is the standard audiogram and the bone conduction curve on a patient with early conduction deafness. Apparently the Rinne is still positive. The 128 tuning fork, however, shows



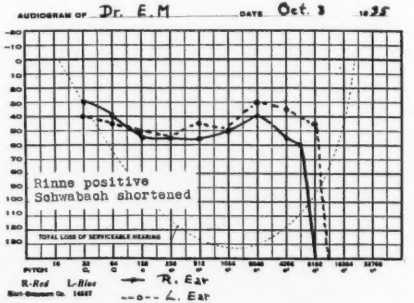
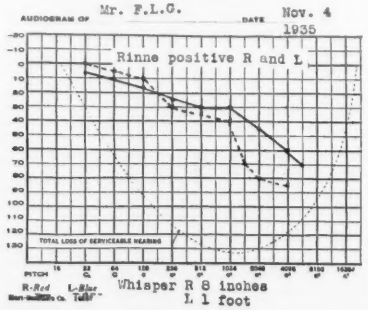


Chart 1. Case of conduction deafness and one of nerve deafness with nearly identical audiograms. A—Audiogram of a patient with conduction deafness. B—Similar audiogram of a patient with primary nerve deafness.


AUDIOGRAM OF Mr. F.L.C. DATE Nov. 4 1935
Rinne positive R and L
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
R-Red L-Blue
Whisper R 8 inches
L 1 foot

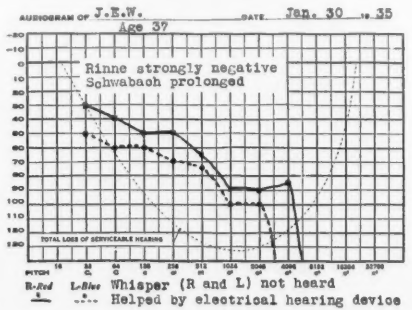

AUDIOGRAM OF J.E.W. Age 37 DATE Jan. 30 1935
Rinne strongly negative
Schwabach prolonged
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
R-Red L-Blue
Whisper (R and L) not heard
Helped by electrical hearing device

Chart 2. Case of nerve deafness and one of conduction deafness with nearly identical audiograms. A—Audiogram of a patient with primary nerve deafness. B—Similar audiogram of a patient with conduction deafness with secondary nerve degeneration.

clearly that the Rinne is negative in this ear. Again there is sufficient bone conduction from the standard receiver to give a false picture of the hearing for low tones in conduction deafness. A pure air conduction curve can be obtained only by holding the standard receiver close to but not touching the ear.

Chart 5.—Now we see that the Rinne is indeed negative for the low tones.

Chart 6.—Here are all three curves, the standard audiogram, pure air conduction and bone conduction, for a patient with nerve deafness and for a second patient with conduction deafness. The latter cannot hear the 32 fork by air, and notice that the pure air conduction curve shows this. By comparing pure air with bone conduction the diagnosis can be made in each patient. It seems as though we had finally succeeded in making the audiometer take the place of the tuning fork tests. There are, however, some serious inaccuracies in the pure air conduction curve.

Chart 7.—This patient has one normal hearing ear and one dead ear following a labyrinthectomy because of meningitis complicating a suppurative labyrinthitis. Notice in the normal ear how far pure air conduction is below the standard curve. This is largely due to the fact that when the canal is occluded by the audiometer receiver a standing wave is set up between the drum membrane and the receiver, increasing considerably the amplitude of vibration of the drum membrane; the external canal, in other words, acts as a sounding box. This involves predominantly the middle and lower tone range.

As soon as there is an air space around the receiver there is an immediate, marked diminution in the loudness of the sound. To obtain an accurate pure air conduction curve the audiometer would have to be recalibrated for this new condition. A second objection is the difficulty in holding the earphone at exactly the same distance from the ear without touching. A third objection is that there is increased diffusion of sound into the outer air which passes around to the better hearing ear, giving a false picture of the hearing in the poor ear, where there is a great difference between the two ears. This, of course, can be eliminated only by masking the good ear with some sort of noise apparatus.

I wish to call attention to the fact that the Schwabach test sums up the hearing for the two ears so that in unilateral nerve deafness, though the other ear be perfectly normal, the Schwabach will be shortened.

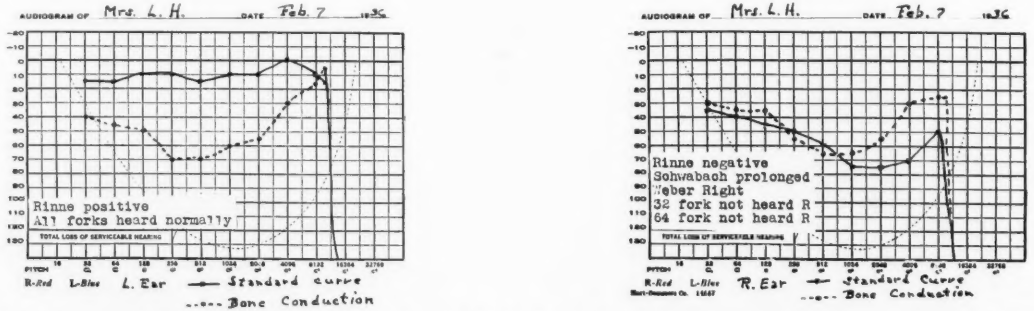

AUDIOGRAM OF Mrs. L. H. DATE Feb. 7 1936
Rinne positive
All forks heard normally
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
R-Red L-Blue L. Ear Standard curve
Bone Conduction
AUDIOGRAM OF Mrs. L. H. DATE Feb. 7 1936
Rinne negative
Schwabach prolonged
Weber Right
32 fork not heard R
64 fork not heard R
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
R-Red L-Blue R. Ear Standard Curve
Bone Conduction

Chart 3. Standard audiogram and bone conduction curve on patient with unilateral conduction deafness. A—On normal hearing left ear. B—On hard of hearing right ear.

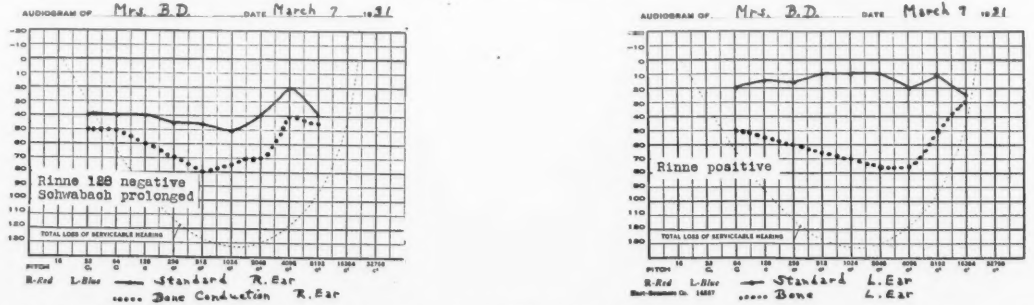

AUDIOGRAM OF Mrs. B.D. DATE March 7 1931
Rinne 128 negative
Schwabach prolonged
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
R-Red L-Blue Standard R. Ear
Bone Conduction R. Ear
AUDIOGRAM OF Mrs. B.D. DATE March 7 1931
Rinne positive
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
R-Red L-Blue Standard L. Ear
Bone L. Ear

Chart 4. Standard audiogram and bone conduction curve on patient with early conduction deafness. A—On right ear with early conduction deafness. B—On normal hearing left ear..

To summarize, the standard audiogram by itself is of no value for diagnosing the type of deafness. It gives a false picture of the hearing for low tones in cases of conduction deafness, and it cannot be used with the bone conduction curve for diagnosis. The pure air conduction curve removes the element of bone conduction, but before it can be used the audiometer must be recalibrated. In all the audiometer tests masking must be used to exclude the good ear where there is a great difference in hearing acuity of the two ears. Finally, and by no means least important, the time required for these audiometer tests is so excessive as to be prohibitive for the practicing otologist unless a technician runs the tests, and one hesitates to rely entirely upon a technician where so much may hinge upon a positive or a negative Rinne, a prolonged or a shortened Schwabach. Moreover, when one uses a complicated mechanical device like the audiometer there are more opportunities for error to creep in than with the very simple, direct tuning fork tests. The element of fatigue, for instance, may be important in making these prolonged audiometer tests inaccurate and unreliable. Bone conduction tests made with tuning forks are simple, quick, direct and accurate, and for ordinary clinical use in the diagnosis of deafness seem to be more practical and reliable.

Concerning the making of bone conduction tests with tuning forks there are a few relevant observations.

Chart 8.—In early conduction deafness the Rinne becomes negative first for the low forks, later for the higher forks. The 128 fork is, therefore, preferable to a higher one for the detection of early conduction deafness.

The Rinne and Schwabach tests are sometimes made by timing the duration of hearing with a stop watch, the fork being left in place until its tone is no longer heard. This method introduces the errors of auditory after-images and auditory fatigue, which make it difficult for a patient to say just when the sound dies out. A more accurate method is to transfer the fork repeatedly from ear to meatus for the Rinne test, and from skull to skull for the Schwabach test. This also removes any error due to differences in the blow administered to the fork.

There is one very important consideration with regard to making the Schwabach test. The prolongation of bone conduction in lesions of the conducting apparatus is due mainly to the exclusion of outside sounds by the conduction lesion so that the inner ear is, so to speak, in a soundproof environment. Knudsen and Jones were unable to

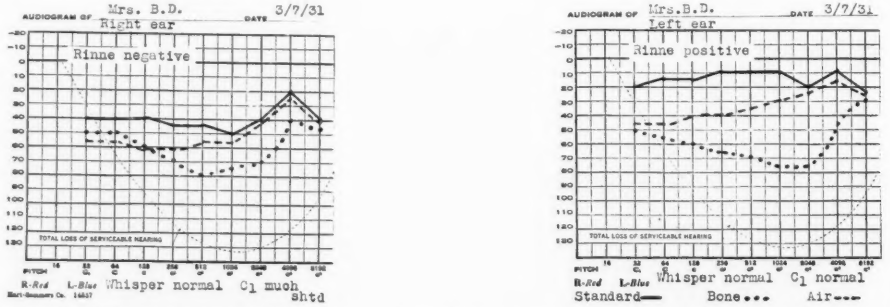


Chart 5. Same patient as chart 4 with additional pure air conduction curve obtained by holding earphone close to but not touching ear thus removing all possibility of bone conduction from standard receiver. A—Same patient as previous with additional pure air conduction curve.

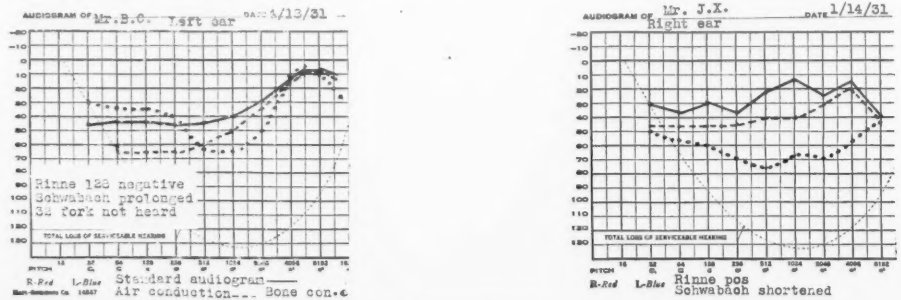


Chart 6. Standard audiogram, bone conduction audiogram and pure air conduction audiogram on patient with conduction deafness and patient with nerve deafness. A—Conduction deafness. B—Primary nerve deafness.

find any prolongation of bone conduction above normal in patients with otosclerosis, chronic catarrhal otitis media, chronic purulent otitis media or middle ear trauma when tested in a soundproof room. It follows that the Schwabach test is of no value in the detection of conduction deafness if made in a soundproof room. It also follows, though I have not found it mentioned before, that the noisier the surroundings the more distinctly will the Schwabach be prolonged in conduction deafness.

Chart 9.—The Schwabach test was made on one patient with conduction deafness, one with normal hearing and one with primary nerve deafness; first in a quiet, inside room which was not soundproof, and then in a fairly noisy room with windows open toward Michigan avenue. Notice that in the quiet room bone conduction was so slightly prolonged in patient A as to be doubtful, while in the noisy room the Schwabach was very definitely prolonged. For practical purposes, the average office is suitable for the Schwabach test. When the test is doubtful, opening the windows and increasing the noise will help to bring out a prolonged bone conduction.

It is generally assumed that in patients with an approximately equal impairment of hearing but with different lesions of the conduction apparatus the bone conduction tests give the same results. Is this true?

Chart 10.—Here we have an acute suppurative otitis media of two days' duration; the whisper is reduced to four inches for the number "44," the Schwabach is prolonged, the Rinne negative and the Weber is heard in the affected ear. Notice the standard audiogram curve and the one for bone conduction with loss for the high tones almost as great as for the low. The 32 fork is only moderately shortened, the 64 fork is only slightly shortened, while the 2048 fork is also slightly shortened.

The second patient has otosclerosis with nearly the same degree of deafness in the poor ear as the previous patient, the whispered "24" being heard at six inches. Here, too, the Schwabach is prolonged, the Rinne negative and the Weber is heard in the poor ear, but notice that the 32 fork cannot be heard at all, the 64 fork is much shortened, while the 2048 fork is only slightly shortened. Notice the standard and bone conduction curves with excellent hearing for the high tones. Corresponding to this, the low pitched sound of "24" is heard at six inches, the higher pitched sound of "66" is heard at two feet, while in the previous patient "66" was heard only close to the ear and "44" was heard at four inches.

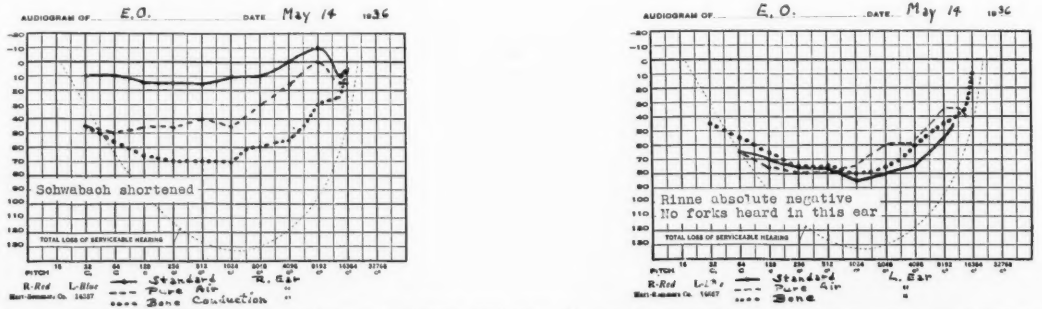


Chart 7. Standard audiogram, bone conduction audiogram and pure air conduction audiogram on patient with unilateral dead labyrinth the result of labyrinthectomy for suppurative labyrinthitis complicated by meningitis. A—On normal hearing ear. B—On dead ear.

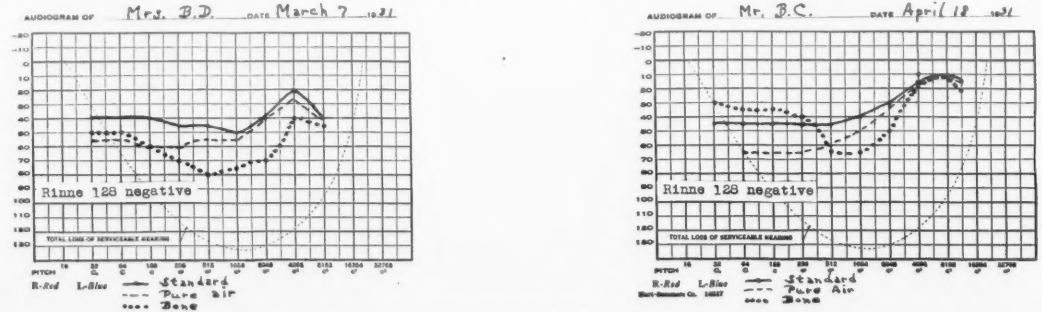


Chart 8. Standard audiogram, bone conduction audiogram and pure air audiogram on patient with early conduction deafness and patient with advanced conduction deafness. A—Early conduction deafness. B—Advanced conduction deafness.

The third patient has an acute catarrhal otitis media of one month's duration, the middle ear cavity being filled with clear, yellow serum shining through the transparent drum membrane. The whisper is reduced to six inches for "24" in the affected ear, an identical degree of deafness with the previous case of otosclerosis, yet the 32 fork in this patient is only slightly shortened, the 64 fork is only very slightly shortened, while the 2048 fork is slightly shortened. Here again the Schwabach is prolonged, the Weber is referred to the affected ear, but the Rinne is still positive. Notice the audiogram curve showing considerable impairment for high tones. One might suspect that there is an element of nerve deafness in this patient and also in the first patient, causing the loss for high tones. This is proved by the fact that only a few minutes after this test was made the drum membrane was punctured and the fluid blown out of the middle ear, with immediate restoration to normal hearing for all tests.

For the fourth patient, hearing tests made two years ago showed normal hearing in the left ear. She now has a large, traumatic rupture of the drum membrane following a domestic altercation. The whisper is reduced to one foot for "24." The 32 fork is not heard at all, and the 64 fork is much shortened, the 2048 fork only slightly shortened. Notice the audiometer curve with relatively good hearing for the high tones and corresponding to this the whispered "66" is heard twice as far from the ear as the "24."

In those patients whose middle ear cavity is filled with fluid interfering with the movements not only of the drum membrane and stapes but also of the round window membrane, there is a loss involving the whole tone scale, the high tones as well as the low tones. The Schwabach is prolonged and the Rinne may be a shortened posi-

CHART 9

SCHWABACH TEST DONE IN A QUIET AND IN A NOISY ROOM

		A Pt. with conduction deafness	B Examiner	Pt. with nerve deafness
Quiet	A fork	51 seconds	45 seconds	12 seconds
inside	c fork	25 seconds	22 seconds	8 seconds
room	a^1 fork	38 seconds	36 seconds	28 seconds
Noisy	A fork	31 seconds	20 seconds	8 seconds
outside	c fork	14 seconds	8 seconds	4 seconds
room	a^1 fork	25 seconds	10 seconds	10 seconds

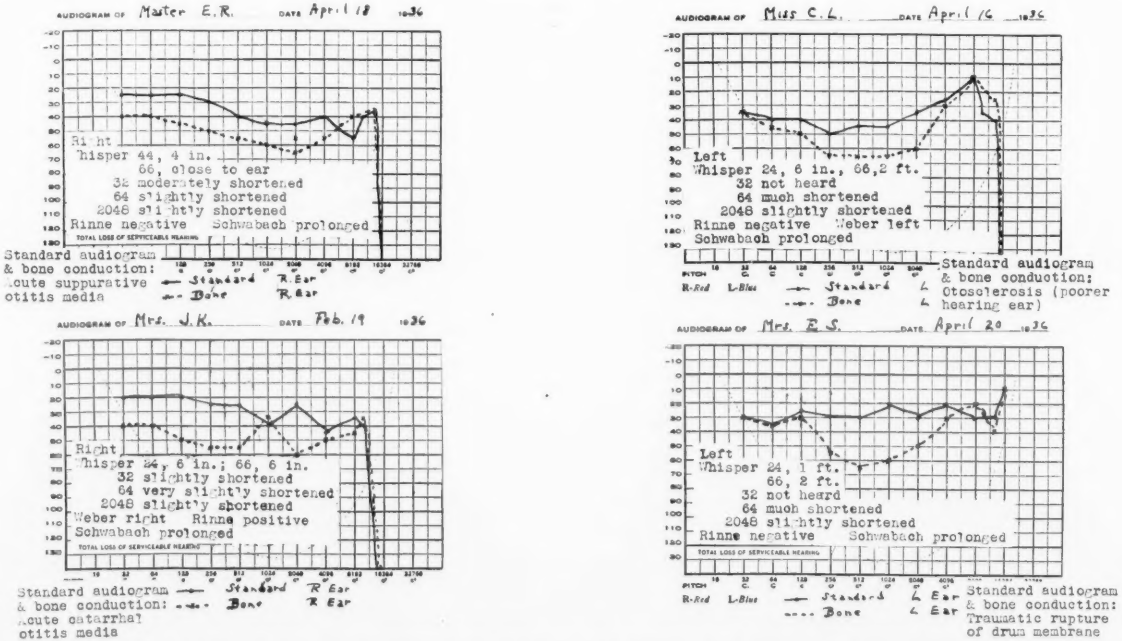


Chart 10. Standard audiogram and bone conduction audiogram on four patients with conduction deafness. The first and third having lesions impeding the movements of both oval and round windows. The second and fourth have lesions involving only the oval window.

tive or equal, instead of negative. In those patients with a lesion involving only the ossicles and stapes, leaving the round window free, the hearing for low tones is greatly depressed, for high tones is hardly affected at all, while the Rinne is strongly negative early in the lesion.

Chart 11.—Five cases of acute catarrhal otitis media seen during the past year are compared with five cases of otosclerosis with approximately the same degree of deafness.

Occlusion of the external canal gives hearing tests similar to cases of acute catarrhal otitis media.

To sum up, different types of conduction deafness give different kinds of hearing tests. Where the movements of the stapes alone are impeded there is a marked loss for the low tones, an early negative Rinne and very little loss for high tones; where the movements of both the stapes and round window membrane are impeded either by an occluded external canal or by fluid in the middle ear there is a more nearly equal loss for low and high tones, while the Rinne may be shortened positive or equal instead of negative.

In all these cases of conduction deafness the diagnosis is usually made with relative ease by means of the tuning fork tests. Before closing I wish to call attention to one type of nerve deafness which is easily confused with conduction deafness because the defect involves chiefly the low tones. I refer to the syndrome of diplacusis and low tone nerve deafness. Since the first three cases were published a year ago I have observed a number of additional cases, three of which I shall briefly mention.

Chart 12.—The first patient, a woman of 40, was first seen in March, 1935, because of high pitched tinnitus in the left ear for seven weeks. Hearing tests showed normal hearing. Two dead teeth were x-rayed and no abscesses found. The diagnosis was possible incipient otosclerosis. Three months later the patient returned because for two weeks the tinnitus had been much worse, with a distinctly jarring discomfort in this ear on exposure to loud noises. Hearing tests showed a low tone nerve deafness with diplacusis for the low forks, which were heard at a higher pitch in the affected ear. The diagnosis of exudative labyrinthitis was made and removal of the dead teeth advised. This was done, one tooth being found abscessed. Within a week the ear symptoms began to improve and soon disappeared. Three months later the patient was tired out and run

CHART 11

HEARING TESTS ON FIVE CASES OF ACUTE CATARRHAL OTITIS MEDIA COMPARED WITH FIVE CASES OF OTOSCLEROSIS WITH SIMILAR DEGREES OF HEARING IMPAIRMENT

ACUTE CATARRHAL OTITIS MEDIA	OTOSCLEROSIS
M. H.—Whisper R 22, 18 in. 66, 6 in 32 fork much shortened 64 fork slightly shortened 2048 fork scarcely heard Rinne positive	Dr. S. L. M.—Whisper L 22, 6 ft. 66, 10 ft. 32 fork not heard 64 fork much shortened 2048 fork slightly shortened Rinne negative
E. R.—Whisper R 22, 1 ft. 66, 6 in. 32 fork moderately shortened 64 fork slightly shortened 2048 fork slightly shortened Rinne equal	Mrs. W.—Whisper R 22, 1 ft. 66, 2 ft. 32 fork much shortened 64 fork moderately shortened 2048 fork normal Rinne negative
C. D.—Whisper R 22, 2 ft. 66, 4 ft. 32 fork moderately shortened 64 fork normal 2048 fork slightly shortened Rinne shortened positive	M. L. Q.—Whisper L 22, 18 in. 66, 2½ ft. 32 fork not heard 64 fork much shortened 2048 fork normal Rinne negative
F. C.—Whisper R 22, 6 in. 66, close to ear 32 fork moderately shortened 64 fork slightly shortened 2048 fork much shortened Rinne not recorded	Mr. N.—Whisper L 22, 6 in. 66, 1 ft. 32 fork not heard 64 fork scarcely heard 2048 fork slightly shortened Rinne negative
A.L.H.—Whisper R 22, 18 in. 66, 2 ft. 32 fork slightly shortened 64 fork slightly shortened 2048 fork slightly shortened Rinne equal	Mrs. B.—Whisper R 22, 1 ft. 66, 3 ft. 32 fork not heard 64 fork not heard 2048 fork slightly shortened Rinne negative
Both windows impeded: All tones affected. Only moderate impairment of low tones. Rinne—shortened positive, equal, rarely negative.	Stapes only impeded: Low tones predominantly affected. Marked impairment of low tones. Rinne—negative.

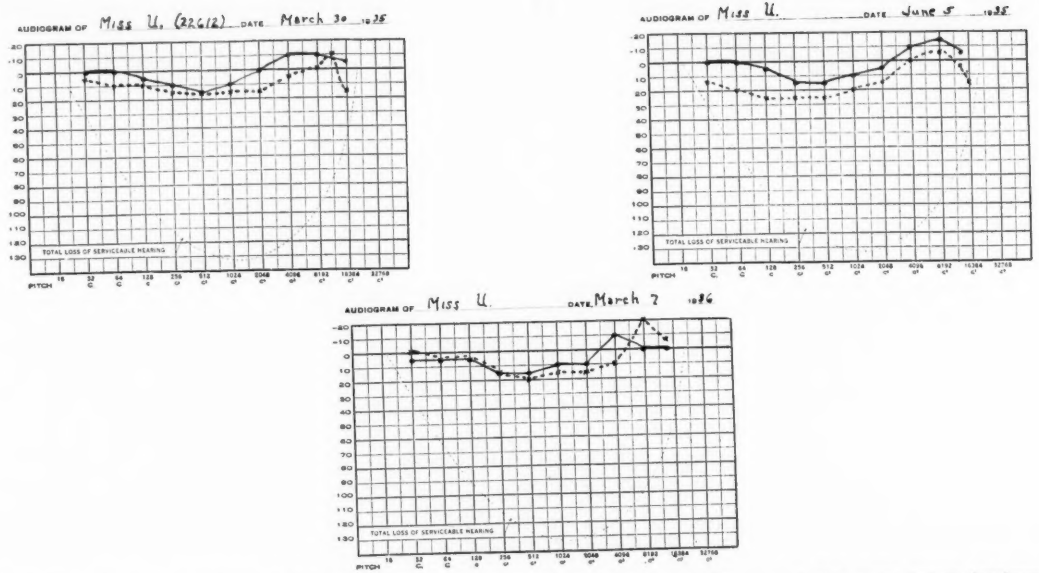

AUDIOGRAM OF Miss U. (22612) DATE March 30 1935
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
AUDIOGRAM OF Miss U. DATE June 5 1935
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
AUDIOGRAM OF Miss U. DATE March 7 1936
TOTAL LOSS OF SERVICEABLE HEARING
PITCH

Chart 12. Standard audiograms of a patient with the syndrome of low-tone nerve deafness and diplacusis: before development of the lesion, during the active process and following removal of the cause (infected tooth). A—Normal hearing at first examination. B—Low tone nerve deafness in left ear with diplacusis. C—Normal hearing; diplacusis largely disappeared.

down and for several weeks the ear symptoms recurred, with three separate attacks of vertigo, each lasting several hours. Since then she has been free from any ear symptoms, the tinnitus has entirely disappeared, and the diplacusis is gone except for the A fork, which is still heard slightly higher pitched in the left ear.

As I pointed out a year ago, the symptom of diplacusis can only be explained by a lesion of the vibrating membrane in the cochlea, such as edema or an adherent, inflammatory exudate weighting down and altering its phase of vibration. It is significant that in every early case of diplacusis that I have seen the tone was always heard at a higher pitch in the affected ear, which is what would be expected if the lesion is an inflammatory change increasing the size and weight of the vibrating membrane. It is also significant that the fork is heard in the affected ear as a jarring, uncomfortable, harsh noise rather than as a pure, pleasant tone. This lesion in the labyrinth is somewhat analogous to iritis in the eye. Both conditions are often due to focal infection, both involve usually one side but later may become bilateral; both are subject to relapses and recurrences and, if the cause can be discovered and removed, both have usually a good prognosis.

Chart 13.—The second patient first consulted Dr. Shambaugh, Sr., in 1917, at which time was observed an old, dry perforation of the right drum membrane and a conduction deafness. The hearing in the left ear was normal. In 1919 she returned because of severe, continuous tinnitus. Suspecting that this might be due to a neuritis of the eighth nerve, removal of a large, infected tonsil stub was advised, but this was not done and the tinnitus gradually subsided. In 1934 severe tinnitus returned about the time of a peritonsillar abscess. The tonsil tag was removed but the tinnitus persisted with gradual impairment of hearing in the good ear. She returned to us in 1935, at which time the tests shown in chart 13 were made. The diagnosis was an exudative labyrinthitis from focal infection and she was advised to have several abscessed teeth removed. This was done, with a rapid improvement in the ear symptoms and a cessation of the tinnitus for about a week followed by a relapse. She noticed that low pitched sounds, including her husband's voice, caused a disagreeable sensation in the left ear, while women's voices did not. For the next two months the hearing in the left ear intermittently improved, then became worse again. The patient was advised to have markedly thickened lateral pharyngeal bands removed as the only remaining probable focus discoverable, but she delayed until one day she had a sudden, severe attack of vertigo with nausea, vomiting and

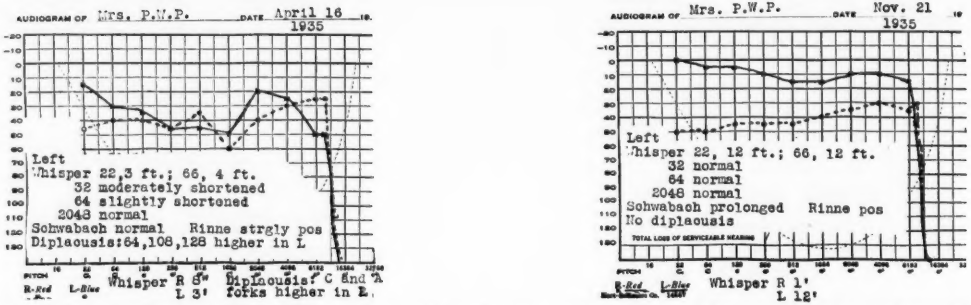


Chart 13. Standard audiograms on a patient with an old suppurative otitis media in right ear and acute exudative labyrinthitis in left ear before and after removal of cause (infected lateral bands). A—Low-tone nerve deafness in left ear with diplacusis. Conduction deafness in right ear. B—Normal hearing in left ear. No diplacusis.

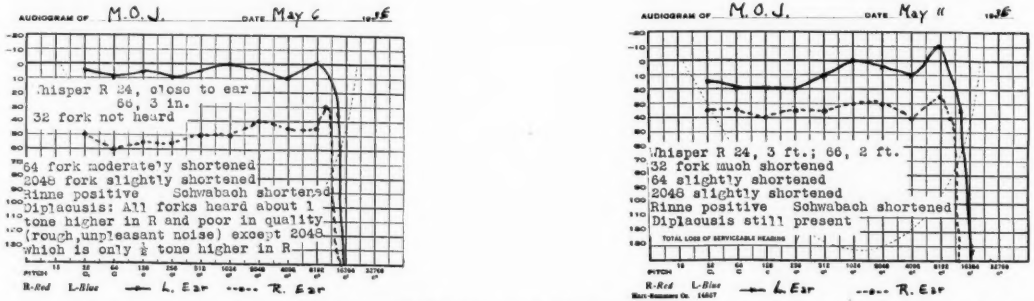

AUDIOGRAM OF M.O.J. DATE May 6
Whisper R 24, close to ear
66, 3 in.
32 fork not heard
64 fork moderately shortened
2048 fork slightly shortened
Rinne positive Schwabach shortened
Diplacusis: All forks heard about 1 tone higher in R and poor in quality (rough, unpleasant noise) except 2048 which is only ½ tone higher in R
L. Ear
R. Ear
AUDIOGRAM OF M.O.J. DATE May 11
Whisper R 24, 3 ft.; 66, 2 ft.
32 fork much shortened
64 slightly shortened
2048 slightly shortened
Rinne positive Schwabach shortened
Diplacusis still present
L. Ear
R. Ear

Chart 14. Patient with unilateral exudative labyrinthitis before and after removal of cause (suppurative ethmoiditis). A—Low-tone nerve deafness with diplacusis, right ear.

ataxia. The lateral bands were removed a few days later, the ear symptoms gradually improved over the succeeding three months until finally the hearing was entirely normal, the tinnitus had entirely disappeared, her husband's voice was no longer unpleasant, and the diplacusis was no longer present. Except for occasional slight ringing in the left ear she has remained entirely free from symptoms for nearly a year. This case brings out the tendency to exacerbations and recurrences in exudative labyrinthitis.

Chart 14.—The last patient of this group complained of tinnitus and deafness in the right ear for one month following a severe cold. There was no vertigo, but he had noticed slight ataxia. The tuning fork hearing tests showed the typical picture of exudative labyrinthitis. Search for a focus of infection disclosed a suppurative ethmoiditis which cleared up after a few Proetz displacement treatments. Following this there was a marked improvement in hearing. Could this diagnosis have been made from the audiograms alone?

Chart 15.—Here are this patient's standard, pure air conduction and bone conduction curves. First of all, the standard curve fails to show the greater loss for low tones because of the element of bone conduction to the opposite better hearing ear. The pure air conduction curve does show the predominant loss for low tones. Secondly, the Rinne is apparently negative, and this can be explained by the fact that the audiometer is not properly calibrated for pure air conduction. Thirdly, there was nothing about the audiometer tests to suggest diplacusis, yet this was easily detected with the tuning forks. I might mention that in testing for diplacusis it is absolutely necessary that the fork be placed at such a distance from each ear that the intensity of the sound is equal, otherwise many patients will confuse intensity with pitch. This adjustment, quickly made with the forks, would be well nigh impossible with the audiometer.

In these patients with exudative labyrinthitis the Schwabach is of paramount importance in differentiating the low tone nerve deafness from conduction deafness. The Rinne, which may remain positive even in severe unilateral lesions, as this last case, is also helpful in ruling out a conduction lesion. Since these patients have normal drum membranes and patent eustachian tubes the diagnosis would be otosclerosis were it not for the tuning fork bone conduction tests. The importance of differentiating this curable form of deafness from otosclerosis is quite evident.

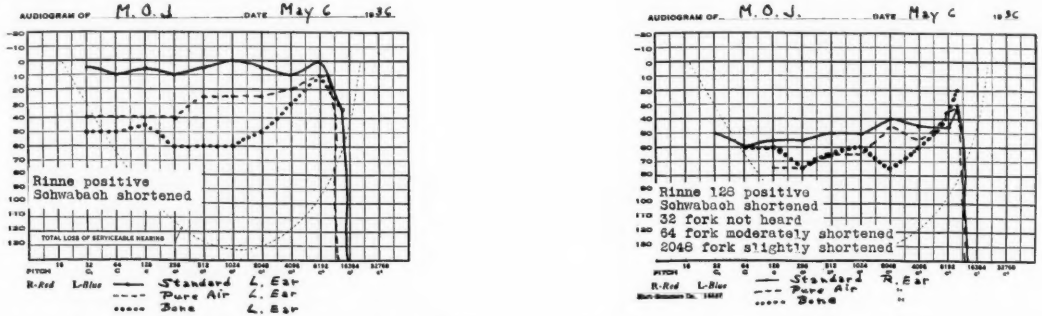


Chart 15. Same patient as chart 14 with standard audiogram, bone conduction audiogram and pure air conduction audiogram showing difficulty of making diagnosis of nerve deafness from audiogram alone. A—On normal hearing ear of previous patient. B—Same on deafened ear showing apparently negative Rinne.

CONCLUSIONS

1. The audiometer cannot be used for diagnosis to the exclusion of the tuning fork tests. The latter have the additional advantage of speed, simplicity and accuracy.

2. The Schwabach test, for the diagnosis of conduction deafness, should not be made in a soundproof room. The noisier the room the more clearly will the Schwabach be prolonged in conduction deafness.

3. Middle ear lesions involving both windows produce more or less uniform loss for all tones; those involving only the oval window produce a marked loss for low tones, very little loss for high tones.

4. The differential diagnosis for low tone deafness from conduction deafness is particularly difficult and very important, and can only be done by the use of the tuning fork tests.

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LVIII

BRAIN TUMORS AND HEARING*

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R. SCOTT-MONCRIEFF, M.D.
D. L. REEVES, M.D.

MONTREAL

The object of this research was to investigate bone conduction acuity in a series of patients suffering from inner ear or proven cochlear nerve lesions, such as acoustic nerve or other intracranial tumors. The patients examined were for the most part from the Neuro-Surgical service, and we are greatly indebted to Dr. Wilder Penfield for placing at our disposal this excellent material.

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*From the Departments of Neuro-Surgery and Otolaryngology, Royal Victoria Hospital, Montreal.

Note—This is a resume of a report of experiments carried out as part of a Symposium on Bone Conduction, for the Detroit meeting of the American Otological Society, 1936.

Much care was exercised in selecting the instruments for testing bone conduction. We were guided in our selection by the report of the Committee on Hearing Tests, of the Royal Society of Medicine, London, 1933. Much valuable assistance was given us by Professor W. Watson, of the department of Physics, McGill University.

A suitable routine of examination was arrived at and was used in all cases except where the preliminary hearing tests had been done before this investigation was undertaken. In so far as possible a complete hearing test was done before and after any operative procedure.

The complete examination consisted of:

(a) A careful history and inspection of the ears, nose and throat.

(b) An audiogram (2A Western Electric Audiometer).

(c) Tests with the audiometer bone conduction apparatus of bone conduction and absolute bone conduction for the notes 256 d.v. and 512 d.v.

(d) Tests with a tuning fork (512 d.v.) of air conduction, bone conduction and absolute bone conduction.

(e) Tests with a von Struycken monochord of air conduction and bone conduction.

(f) A vestibular examination.

A standard length of vibration of the tuning fork was obtained by using a metal clamp which was removed suddenly, it having slightly compressed the prongs. The vibration time was recorded in seconds.

When there was a difference between the hearing in the two ears, the nontested ear was masked with either a continuous current of air, an electric buzzer or with a Bárány noise apparatus.

The patients examined fall into five groups for descriptive purposes:

Group 1. Thirteen cases were examined before and after encephalography and ventriculography to determine the effect of possible changes of intracranial pressure upon the hearing. A change in hearing was not noted in any case.

Group 2. Fourteen patients had varying quantities of cerebral tissue removed from different areas, usually because of epilepsy or

trauma. As noted by previous observers, it was found that large portions of cerebral tissue may be removed from one side without causing any permanent hearing damage.

Group 3. There were six cases of cerebellar tumor. When the lesion was confined to the cerebellum there was very little change in hearing.

Group 4. In nine cases of cerebral tumor there was no instance of hearing loss attributable to the brain lesion.

Group 5. There were four cases of perineural fibroblastoma of the eighth nerve and one of the fifth nerve. Usually there was almost complete deafness on the involved side but it required masking to demonstrate the true condition. In one case, where the eighth nerve was not completely damaged, the hearing actually improved post-operatively on the diseased side.

Group 6. There were five miscellaneous cases in this group, including an eighth nerve section for Ménière's Syndrome, and one case of third ventricle tumor which showed a loss of high tones after removal of the tumor.

CONCLUSIONS

Of the three methods used to test bone conduction (the audiometer B.C. attachment, the tuning fork and the monochord), one did not prove any more reliable than the others.

Whether bone conduction was tested on the audiometer for the note 512 d.v. or 256 d.v. made very little difference.

In four patients, each of whom apparently had one totally deaf ear, the bone conduction was greater in the poorer hearing ear.

The Weber test proved to be very misleading.

The monochord served as a valuable check upon the audiometer, but it is difficult to mask out its effect on the nontested ear. The continuous current of air proved to be the most effective masking apparatus for the monochord.

Our experiments would suggest that the most accurate method of testing hearing at present is to combine the use of the audiometer, the tuning forks and the monochord for testing both air and bone conduction and that masking the nontested ear may be an essential part of the test.

During this investigation fifty-two patients were examined. In the complete report a short summary will be given of each case.

LIX

IV. REPORT OF THE COMMITTEE ON METHODS OF TESTING THE HEARING BY BONE CONDUCTION*

To the Members of the American Otological Society:

The committee on methods of testing hearing by bone conduction respectfully submits the following report for your consideration.

INTRODUCTION

In the letters of invitation to serve on this committee it was termed "Committee on Standardization of Bone Conduction Tests." The members of the committee have changed the name to "Committee on Methods of Testing Hearing by Bone Conduction," because this designation better describes the content of the report we are submitting.

Standardization implies (1) that all tests other than those recommended should be abandoned, and (2) that abundant evidence is available for the support of the judgments. Actually, our knowledge is so incomplete with respect to the normal and the pathological functioning of the numerous structures concerned with the perception of sound that only guesses can be made as to the potential value of different bone-conduction tests. Whereas it is true that we do not know how to interpret the information from certain bone-conduction tests that have been proposed in the literature, evidence is lacking for the rejection of any test on the grounds that the observations themselves have no diagnostic significance, provided the test is properly made.

On the other hand, there is abundant evidence for the support of three opinions: (1) that bone-conduction tests made without regard to proper precautions yield incorrect observations; (2) that but few otologists make bone-conduction tests in conformity with any of the more precise methods that have been described in the literature; and (3) that the problems presented by patients with impaired hearing cannot be solved by the use of unreliable methods of clinical examination. The solution of these important problems is of interest to every otologist; and, in the last analysis, they can be solved only by the utilization of clinical studies. Animal experimentation, at best, can only furnish clues. The better the clinical observations and

*The members of the committee are: Drs. C. C. Bunch, Harvey Fletcher, Edmund P. Fowler, Stacy R. Guild (chairman), Douglas Macfarlan, A. G. Pohlman.

the greater the number of dependable observations, the better will be the chances of learning how to diagnose lesions that now pass unrecognized.

Therefore your committee believes that it can best contribute to the symposium today and to the eventual solution of some of the problems of hearing by bone conduction through placing the emphasis of its report (1) upon the known causes of incorrect observations, and (2) upon simple methods of taking the precautions necessary for the avoidance of errors. The methods suggested in the report are practicable for adoption by otologists generally, and bone-conduction tests made with the observance of these precautions should yield accurate clinical information that is of value in the solution of the problems presented by patients with impaired hearing.

At the present time it is impossible to correlate and thus utilize most of the published data on hearing by bone-conduction, for the reason that the same words are frequently used to record what are fundamentally different facts. For instance: "shortened bone-conduction time" may mean only that the examination room was noisy, that a defective tuning fork was used, or that the fork was not handled properly. Certainly a common language is needed, whether or not its use leads to any advance in knowledge. The first requisite for a common language is a reasonable uniformity of method, and the first step in the attainment of uniformity of method is the avoidance of inaccurate methods. The committee believes that otologists will at least understand each other better if, in the making of whatever bone-conduction tests they do make, they observe the recommended precautions. This report will have served a useful purpose if it leads to the securing of more accurate data by otologists generally.

Hearing by air conduction and by bone conduction are so intimately related that it is necessary to include in this report some consideration of methods of testing by air conduction.

The body of the report* is presented under five headings:

1. Suitable sources of sound for bone-conduction tests.

2. The effect of room noise upon the accuracy of tests of hearing.

*The facts set forth and the recommendations made in this report are, for the most part, based on the experiences of individual members of the committee. Bibliographic references are omitted from the report because of the large number of articles that would have to be cited were any attempt made to cover this phase of the topic adequately.

3. The use of masking in clinical tests of hearing.

4. Special points about the use of tuning forks.

5. Special points about the use of audiometers with bone-conduction receivers.

SUITABLE SOURCES OF SOUND FOR BONE-CONDUCTION TESTS

Sources of sound which produce only one intensity, such as watches or acumeters, are of little value for quantitative studies of hearing by bone conduction. Those patients who do hear the sound from such a source cannot be reliably divided into smaller groups on the basis of the acuity of hearing, because the subjective impressions of different patients as to how well a sound is heard cannot be accurately compared.

For threshold tests. In order to obtain reliable quantitative measurements of the acuity of hearing by bone conduction it is necessary to use a source of sound that produces a wide range of intensities, so that the threshold between "heard" and "not heard" can be determined for each patient who hears the sound. This requirement is met by only two types of sources of sound. The first type includes all objects whose intensity of vibration decreases gradually from some initial intensity to zero: the most commonly used sound-sources of this type are tuning forks. The second type of sound-source includes all objects whose intensity of vibration can be maintained indefinitely at any chosen level within its range, or altered as desired: for practical purposes this type of sound-source is limited to the so-called bone-conduction receivers actuated by some form of electric audiometer.

Accurate data obtained with either tuning forks or bone-conduction receivers are of value in the study of hearing by bone conduction. The groups into which patients are divided by good tests with one tuning fork of a given frequency can, by cross-calibration* of instruments, be made comparable to the groupings into which patients are divided by good tests with another tuning fork of the same frequency or by the use of a bone-conduction receiver. It should be emphasized, however, that for the data obtained by one otologist to be of value to others, it is necessary that, whatever the instruments used, the tests themselves be done in such manner and under such conditions that the observations obtained are, for the method employed, a true measure of the acuity of hearing. Many

*Methods of calibration are considered in the section on "Special points about the use of tuning forks."

observations have been recorded as measurements of hearing acuity that contribute nothing toward the solution of hearing problems because they are incorrect for one or more of the following reasons: noise in the examining room introduced a factor of unknown size; the examiner used his own hearing as the standard of comparison without knowing how much his own hearing was impaired at the time; a variation in striking the fork or in holding it, either before the meatus or against the head, affected the observations; the audiometer was off of correct calibration for some reason, such as accidents to receivers, loose connections, partially exhausted batteries, etc.; the patient actually heard the sound with his other ear, either by air conduction around the head or by bone conduction through the head. This is but a partial list of the items that may affect the accuracy of data from hearing tests.

There can be no question that the ideal otological examination should include threshold determinations, for both air and bone conduction, of all frequencies, from the lowest to the highest that can be heard. Practically, this ideal is more difficult of attainment for bone-conduction tests than for those by air conduction. The use of the higher frequencies for bone-conduction tests is limited by the fact that, for all tuning forks and many bone-conduction receivers, people with normal hearing can perceive the sound by air conduction when the source is at threshold intensity for hearing by bone conduction and is in the position used for the bone-conduction tests. For the frequencies and the instruments for which this condition obtains it is difficult, and sometimes impossible, to determine accurately the normal threshold for bone-conduction. The use of the lower frequencies for bone-conduction tests of hearing is limited by the factor of tactile sensitivity. It is difficult to make accurate bone-conduction acuity tests if the patient can still feel the vibrations when the source of sound is at the threshold for his hearing. For this reason objective methods cannot be used for the determination of impaired hearing by bone conduction for frequencies in the region of 128 d.v. Many patients with an impaired bone-conduction acuity can feel the vibrations of a 256 d.v. source when they cannot hear the sound.

These limitations to the use of tuning forks for bone-conduction tests have been recognized for several decades, and it was for these reasons that Politzer recommended the 512 d.v. fork and Bezold the 435 d.v. fork as the most suitable for bone-conduction tests. Careful attention should be given to these same limiting factors in the selection and in the use of bone-conduction receivers. Even at fre-

quencies as low as 435 and 512 d.v. some of the larger tuning forks and some bone-conduction receivers radiate so much sound into the air that they are not well suited for use in testing bone-conduction acuity. Your committee agrees with the view that steel forks of the frequency of 435 or 512 d.v. are the forks of preference for bone-conduction tests when the acuity for only one frequency is examined. But the committee urges that, because of the probable value of the additional information, all otologists make bone-conduction acuity tests over as wide a range of frequencies as is possible with the instruments available to them. For use in the evaluation of the data from bone-conduction tests with the higher and the lower frequencies, we also urge that for each patient careful notes be made as to whether or not he could hear the sound by air conduction or could feel the vibrations of the source of sound.

Other facts that must be taken into account with tuning forks or with bone-conduction receivers, if dependable observations are to be secured, are considered in the respective sections on the use of these two sources of sound.

For tests above threshold. Until recently all hearing tests have been made for one or both of two purposes: to obtain information that may aid in diagnosis, or to obtain information of possible value in the study of the physiology of hearing. There is also a third reason for examining the hearing function; namely, to obtain a proper basis for an intelligent opinion as to the best hearing-aid for the patient to secure.

The sounds of most importance to the patient, both socially and economically, are conversation and noises. He wishes to be able (1) to understand the conversational voice clearly and (2) to hear many other sounds distinctly, to enable him to escape danger, do his work properly, etc.

Several papers have appeared in the otological literature on methods of determining the characteristics that an ideal hearing-aid for any particular patient should have. The methods proposed have all been based on determinations of threshold acuities. Your committee wishes to call to your attention that, theoretically at least, threshold acuities for pure tones do not necessarily furnish as good a basis for predicting the ability of a patient to understand speech or to hear noises clearly with any given type of hearing-aid as would actual tests with voice and noises at the intensity levels at which they are clearly heard by normal people. While this information might be of no assistance in the establishment of a diagnosis, it would be a

real aid to the patient and might prove to be of great value scientifically.

There is only a meagre store of objective knowledge with respect to hearing at intensity levels well above threshold, even for sounds transmitted to the ear by air conduction. It has become increasingly evident in recent years that all of the problems of the physiology of hearing cannot be solved by correlations of structural and other facts with data for the thresholds for hearing. This report is not the proper place for the elaboration of this topic; and your committee wishes only to call attention to the facts and to some possibilities. Now that bone-conduction receivers are available it is practicable for otologists to devise and to make tests of the hearing for voice, music and noises by both air and bone conduction at various known intensity levels above the patient's threshold. The accumulation of such data for many patients with impaired hearing should prove of real value in the solution of some of the problems that remain unsolved.

THE EFFECT OF ROOM NOISE UPON THE ACCURACY OF TESTS OF HEARING

If a patient's hearing is impaired to such an extent that he cannot hear the room noise clinical tests of equal accuracy can be made in either a sound-proof or a noisy room. On the other hand, if a patient's hearing is such that he can hear the room noise, the masking effect of this noise impairs his acuity of hearing for the test sounds and therefore the accuracy of the clinical tests by both air and bone conduction. We do not know with certainty whether this is due to interferences set up in the middle ear, the inner ear, the central nervous system, or in all of these parts of the organ of hearing. We do know, however, that the louder the noise, the greater the interference with the hearing.

Therefore, when absolute standards are used, such as the zero reading of an audiometer or the number of seconds that a fork is heard after being struck a standard blow, the data obtained in a noisy room are often not the true measure of the hearing acuity, because the calibrations are for quiet surroundings. In fact, room noise frequently causes failure to detect early stages of deafness, because patients with but slight degrees of hearing impairment cannot be differentiated from those with normal hearing by study of the records of examinations made in noisy surroundings. The exact degree of impairment depends upon the relations between the frequencies of the components of the room noise and those of the test sound, and upon other factors, none of which require discussion in this report.

Practically, it is much easier to avoid the factor of room noise than to determine the proper allowances that should be made for its effect upon each test.

When tests are made by comparison of the patient's hearing with that of the examiner, accurate data can only be secured by also making allowances for the masking effect of the room noise on the examiner and for the examiner's own hearing impairment. Detailed directions for making the latter allowance, for tests in quiet surroundings, are given in the recent report by our British colleagues, which report should be consulted by those interested in this method of testing hearing.

A sound-proof room large enough to provide ideal conditions for tests of hearing acuity is expensive to construct. In most office buildings the only rooms that are satisfactorily quiet for making reasonably accurate hearing tests are inside rooms that do not have windows or doors toward the hallway and that do not have water pipes behind the walls. When such a room is not available the committee recommends the construction of a so-called sound-proof booth within the quietest room that is available. The committee recommends also that the walls of this room or booth be of hard material, because many patients are so disturbed by the absence of reverberation in a padded room that they do not cooperate well in the tests.

THE USE OF MASKING IN CLINICAL TESTS OF HEARING*

Why use masking? Many data recorded as to the hearing of one ear of a patient are, in reality, data as to the acuity of hearing of the other ear for the source of sound when at a greater distance than usual. The sound may have reached the other ear by air conduction around the head or by bone conduction through the head. Such observations have neither diagnostic nor scientific value; they are, in fact, a positive menace to the advance of knowledge.

In order to measure the hearing acuity of the poorer ear of a patient it is necessary first to exclude the possibility that the test sounds are heard by the better ear. Occlusion of the external auditory meatus is useful only for the exclusion of air-conducted sounds, and then only when the difference in acuity between the two ears is small. For bone-conduction tests the only practical method for the exclusion of the better ear is to mask it with other sounds: thus the threshold

*The topic of masking for air-conduction tests was ably presented before this Society five years ago, in a paper by Dr. Bunch. The committee believes that all who undertake for the first time to use masking will profit by the careful study of his detailed presentation.

of the better ear for the test sound can be impaired sufficiently to insure that the data obtained are actually for the poorer air.

Which ear should be masked? It is easy to determine which ear has the better hearing for air-conducted sounds, and therefore which ear should be masked for tests of air-conduction acuity. But for bone-conducted sound it is not always easy to determine which ear has the better hearing. The statements of the patient as to lateralization of the sound from midline points of the head should of course always be recorded, but they do not form a sufficient basis for the selection of the ear to be masked during tests of bone-conduction acuity. One of the unsolved problems of hearing is why many patients with equally good hearing in both ears do lateralize sounds from the midline to one ear. Aside from the subjective character of lateralization there must be, in many instances, an underlying but unrecognized basis of structural differences, such as asymmetries of the head, etc. Such differences offer the only rational explanation that has been advanced for the not uncommon observation that some patients lateralize sound differently from different midline points of the head, and for the more rare instances of lateralization to the opposite ear from the mastoid region by patients who do not lateralize sounds from the midline points.

For the above reasons, and because the entire head actually vibrates during all tests by bone-conducted sound, your committee recommends that, as a routine practice, for all bone-conduction acuity tests each ear be masked while testing the opposite ear. For many patients this is the only method by which the better ear for bone conduction can be determined. Furthermore, the use of masking frequently reveals significant differences between the two ears that were not suspected from the same tests made without masking. Until one has used masking routinely, he does not realize how often such differences are present.

How can one ear be masked? The ideal in masking is the exclusion of all hearing by one ear without in any manner affecting the acuity of the other ear. For hearing tests there is no method that even approaches the ideal, so easily attained in testing vision, because sound waves penetrate where light does not. In strongly recommending that all otologists use masking, the committee is fully aware of the fact that for some patients the acuity of the ear being tested is more or less impaired by the masking sound applied to the other ear. However, from the experiences of the individual members of the committee and of others, we are thoroughly convinced that the infor-

mation gained by testing with the aid of proper masking is far more valuable than is that which can be obtained without masking.

For both air- and bone-conduction tests the masking sound must be introduced into the ear by air conduction. Bone-conducted sound is not suitable for masking purposes because both ears are always stimulated by this method. On the other hand, air-conducted sounds that are loud enough to mask one ear satisfactorily may reach the other ear by a bone-conduction route. For this reason and because masking may be in part due to some form of interference within the central nervous system, the masking sound should be of the minimum intensity that excludes perception of the test sound by the masked ear. The intensity needed for this purpose differs for different patients, and frequently for the two ears of the same patient.

Therefore the apparatus used to produce the masking sound must be adjustable as to intensity. This basic requirement is not met by the apparatus most commonly used for masking; viz., the Bárány Lärmapparat. This instrument is useful only when one desires to mask an ear to the point where even loud shouted voice cannot be heard: furthermore, the sound made by the Bárány Lärmapparat is so intense that in many patients the acuity of the opposite ear is at the same time much impaired.

Mixed sounds, or noises, are better for the purpose that an otologist uses masking than are pure tones. Theoretically, the ideal sound to use for masking would be an even mixture of all frequencies that can be heard, from the lowest to the highest. Such a mixture of frequencies is not produced by any apparatus that is yet available for masking purposes. Fortunately, experience has shown that satisfactory masking for the low and middle range tones, the ones of most importance to the patient, can be accomplished by several different methods, none of which produce the theoretically ideal masking sound. Experience has also shown that for most patients the thresholds for test sounds are but little affected by moderate increases of the intensity of the masking sound above the theoretical minimum requirement. This fact eliminates, for practical purposes, the necessity of using a masking apparatus that can be calibrated as exactly as can a good audiometer or a good tuning fork. However, it does not eliminate the requirement that a masking apparatus, to be satisfactory for daily work, must be adjustable as to the intensity of the sound produced.

Methods of producing satisfactory masking sounds. Four simple, practical and satisfactory methods of masking, with each of

which one or more members of the committee have had extensive experience, are described below: all but one of the apparatuses are relatively inexpensive to build.

Wherever a 60-cycle alternating current is available, a masking apparatus can be made by the use of a step-down transformer, such as is used for doorbell circuits or for electrical toys, an ear-phone, and a rheostat such as is used for the volume-control of the sound from the loud-speaker of a radio receiving set. Depending upon the particular receiver used, it may or may not be necessary also to put a fixed resistance coil in the output circuit in order to make it possible with the adjustable resistance (the rheostat) to produce the desired range of intensities of the masking sound. The entire apparatus, including the parts named, switches, wiring, plugs, and the labor of an electrician, should not cost more than seven to ten dollars. The parts can be compactly assembled, so that the equipment is easily portable, thus making it available for use in the home as well as in the office. The resultant sound is not a pure tone of the frequency of the alternating current of the house-circuit, but it also contains other elements introduced by the imperfections of the parts used. While not ideal, this inexpensive equipment is much more satisfactory for masking purposes than is a Bárány Lärmapparat.

Another method of building a masking apparatus is to pass the direct current from three dry-cell batteries of the doorbell type, connected in series, through a convertor of the vibratory kind that is used in automobile radio sets. Beyond the convertor the construction is exactly as for the apparatus using alternating house-current, except that it is not necessary to use a step-down transformer in order to get a suitable voltage for the ear-phone. The entire equipment, including batteries, can be assembled in a small wooden box. If a panel switch for turning on the current, a jack for the ear-phone connection, and the knob of the rheostat are mounted in the lid of the box, the apparatus is convenient to use and is easily transported from place to place. The total expense need not be more than about eight dollars. It is only necessary to replace batteries about once a year, even though the apparatus is used several times daily. The resultant sound is more complex than is that produced by the first type of apparatus, and is therefore rather better as a masking sound.

The complex sound produced by a Western Electric Co. 3-A audiometer, which was designed for an entirely different purpose, may also be used for masking. Because of its relatively high cost the committee believes that but few otologists will choose this means of masking: however, to those who do possess this instrument the com-

mittee recommends it as a practical means of producing a satisfactory masking sound. Experience with this instrument has shown that the range of intensities from 65 db. to 95 db. above normal threshold suffices for almost all masking purposes.

The same facts that determine this to be the range of intensities of the sound from the 3-A audiometer that are needed for masking purposes can be utilized, during the construction and calibration of a masking apparatus such as one of the types described above, for selecting the proper amount of fixed resistance to use in order that the rheostat will permit adjustments of intensity over the desired range. Tests have shown that, at the intensity of 65 db., masking of one ear of a normal individual does not cause impairment of the thresholds of the other ear, provided a sponge-rubber ear-piece is used on the receiver. The sponge-rubber ear-piece confines the sound to the masked ear and also reduces the intensity of vibration transmitted to the bones of the head. The equivalent intensity for other masking apparatus can be approximated closely enough for practical purposes by the experimental determination of the loudest sound that does not affect the thresholds of normal young adults. At the intensity of 95 db. the sound from the 3-A audiometer impairs the hearing of the masked ear so much that loudly shouted voice at the distance of one foot is not heard, provided the other ear is totally deaf or that it is also effectively masked. This is, for practical purposes, the greatest intensity that any masking apparatus needs to be capable of producing.

For all tests in which masking is accomplished by the use of equipment such as described above, the intensity of the masking sound that is used should be recorded. Since the purpose is not to measure the acuity of hearing for the masking sound but to exclude perception of the test sound by the masked ear, it is sufficient to utilize the approximate calibration given by several arbitrary divisions of the dial of the volume-control rheostat between the settings at which the apparatus produces the minimum and the maximum intensities desired. Thus, even though the gradations of intensity are not exact, it is possible to follow accurately the progress of individual patients because the same intensity of masking can be used at each visit.

Blowing a stream of air across the external auditory meatus is the simplest method of satisfactorily masking one ear while testing the other. A practical method of doing this without the aid of an assistant is to use the ear-pieces of a stethoscope attached by rubber tubing to a source of compressed air. When only a small stream of

air is flowing a hissing sound is produced by the escape of the air between the ear-piece of the stethoscope and the walls of the external auditory canal. An optional method of producing a similar sound is to permit the air to escape through a small hole in the rubber tubing; in this case the stethoscope simply serves to conduct the sound to the ear. With either method of producing this type of sound for masking, its intensity can be adapted to the requirements of individual patients by adjustments of the valve in the air-line pipe, but the intensities actually used cannot be determined for record in terms of decibels or of any other units. For this reason it is impossible, by this method of masking, to duplicate conditions in successive examinations.

When masking is not used the examiner has no choice but to accept the statement of the patient as to which ear hears the sound. The purpose of masking is to enable the examiner to use objective methods in the determination of the hearing acuity of the poorer ear as well as of the better ear. Therefore the determination of the proper intensity of the masking sound must also be made by objective methods: it is not sufficient to accept the word of the patient that he no longer hears the test sound in the ear being masked. Any otologist, after a little experience with any one of the methods of producing masking sounds that are described above, can quickly determine the proper intensities to use with different patients. Therefore detailed directions for the use of each method with each of the different types and degrees of impaired hearing are not included in this report. *The important thing is to use masking and to use it as a routine procedure, whether the hearing acuity is examined by means of tuning forks or by means of audiometers.*

SPECIAL POINTS ABOUT THE USE OF TUNING FORKS

It is well known that tuning forks of the same frequency, and even of the same make, differ greatly from each other in the maximum loudness of the sound that can be produced by them, in their rates of damping, in the total length of time that they can be heard after being struck identical blows, in the acoustic energy given off by the prongs relative to that given off by the stem, and in other ways that can be measured. The importance of using standard blows to activate tuning forks and the need for calibrated forks have been emphasized repeatedly in our literature. But in recent years relatively little has been written or said about an equally important point with respect to the use of forks for bone-conduction tests; namely, the handling of the fork after it is activated. Therefore this topic is considered first.

Technique of handling fork after it is struck. The energy of a vibrating tuning fork is dissipated through four principal routes: (1) heat due to internal friction, (2) acoustic energy transmitted to the surrounding air from prongs and stem, (3) acoustic energy transmitted from the end of the stem to the head of the patient or of the examiner, and (4) acoustic energy transmitted from the stem into the fingers. For any particular fork the rates of dissipation of energy via the first two routes remain fairly constant under all ordinary conditions of hearing tests, but the way the fork is used causes marked variations in the rates of dissipation of energy via the last two routes. The energy of the fork is being dissipated less rapidly when the fork is held before the meatus for the testing of air conduction than when its stem is being pressed against the head for the testing of bone conduction. Also, the mere act of gripping the stem more firmly in order to press it against the head increases the rate of loss of energy from the fork.

It does not require the use of special apparatus to demonstrate the truth of the above statements, since the total number of seconds that a fork is heard can be made to vary over a wide range by deliberate variations in the technique of testing after the fork is activated. For example: if, after striking the fork, the examiner at once places the stem against the head and holds it there steadily until the sound is no longer heard the time is much less than if the examiner, after striking the fork, waits 15 or 20 seconds before first placing the stem against the head and then uses an intermittent pressure until the sound is no longer heard. And an intermediate value for bone-conduction time will be obtained if the examiner alternately places the fork against the head of the patient and against his own head; or if the examiner, beginning immediately after striking the fork, alternately places it against the head and holds it before the external meatus.

Besides causing differences in the time that the fork is heard by bone conduction, the different methods of handling the fork for the bone-conduction tests also cause differences in the total time that the fork is heard by air conduction. Furthermore, the length of time the fork is heard by air conduction after it ceases to be heard by bone conduction is appreciably influenced by the technique used during this part of the test: for instance, the difference between air-conduction time and bone-conduction time is greater if the fork is simply held before the meatus until the patient ceases to hear it than if alternate transfers to the mastoid region are continued. A frequent practice in examining patients already known to have bone conduction

better than air conduction is to hold the fork before the meatus until it is no longer heard by air conduction before beginning the testing of bone conduction. If the same patient's bone-conduction acuity were tested by holding the fork steadily against the head from the time the fork is struck until it ceases to be heard, it would appear to be much poorer than when tested the other way.

With each of the above techniques, and with other methods of handling forks, accurate observations can be obtained, provided that the fork is calibrated for the method by which it is used. The really serious objection to the use of such diverse methods of handling tuning forks is that it is exceedingly difficult, and frequently impossible, to compare the data published by different otologists, even when good forks have been used in quiet surroundings and with proper masking.

From the practical standpoint of the difficulties of cross-calibration, the only technique that makes possible reliable comparisons of the data from groups of observations is that of alternate placements at regular intervals, beginning immediately after the fork is struck and continued until it ceases to be heard by either air or bone conduction, whichever is the more acute. This technique results in an "over-all" rate of dissipation of energy that is reasonably constant. If, for instance, the stem of the fork is held against the head for two seconds out of each five seconds, then the rate of dissipation of energy is the same for each of the five-second periods that the fork can be heard, although energy is lost at different rates during the respective parts of each five-second period of time.

Therefore the committee recommends that all otologists routinely use the technique of alternate placements of the fork. The particular period of alternation that is used is not important, so long as the calibration of the fork is for the period used. The committee suggests that the total cycle of each alternation should not exceed five seconds for the lower frequencies and three seconds for the higher frequencies: thus the "end-point" error is brought within the probable error of other factors that affect all tests made with tuning forks.

Activation of tuning forks. Standard blows. For rough qualitative tests of hearing almost any method of activating a tuning fork suffices, but for exact tests of the acuity of hearing it is essential that for each test made the same intensity of sound be produced.

The idea is prevalent that a fork, after being struck a blow harder than is necessary to produce vibrations of maximum amplitude, settles down to the same maximum within a second or two, and

that therefore it does not matter how hard a fork is struck, so long as it is hard enough. Up to a certain limit, which differs for each individual fork, this is true. But the danger in thus activating forks arises from the fact that blows that are too heavy for the particular fork bend the prongs beyond the elastic limit of the metal and permanently damage the fork. As a result the tests made with a fork before it has been thus damaged are no more comparable with those made afterward than are tests made with an entirely different fork. Furthermore, after a prong has once been bent beyond the elastic limit of the metal, it usually bends yet further each time that a hard blow is struck. Therefore the successive tests made after the time of the first bending cannot be reliably compared even with each other. For this reason the committee urges all otologists to exercise extreme care that their tuning forks are not struck too heavy a blow even on one occasion.

The ideal blow for the activation of any tuning fork is that which causes the prongs to vibrate at the maximum amplitude that does not damage the fork. Blows that are too light do not cause a fork to produce its possible maximum intensity of sound, and thereby limit the usefulness of the fork for the testing of deafened patients. By any of the manual methods it is impossible to strike identical blows each time a fork is used. Numerous special apparatuses have been devised for striking forks mechanically identical blows: most of them do not, for all forks, produce the maximum amplitude of vibration; and all of them are cumbersome to use, as compared to manual striking of a fork. One of the simplest, and, so far as the committee knows, the most practical method of striking forks a standard blow, utilizes the force of gravitation. A metal rod, pivoted at one end, is permitted to swing from the horizontal to the vertical position before striking the tuning fork held in its path. The rod is covered with a length of rubber tubing in order to prevent damage to the surface of the fork and to minimzie the production of overtones.

Damaged and otherwise unsuitable forks. The reasons for recommending steel tuning forks of the frequencies of 435 or 512 d.v. for bone-conduction tests have been stated in the section on suitable sources of sound (*q.v.*).

The inner surfaces of the prongs of all good tuning forks are parallel to each other when the fork is not vibrating. If the prongs of a fork are not parallel, either the fork was poor originally or it has been damaged by the use of heavy blows: the reasons why such

a fork is not suitable for hearing tests are given in the preceding subsection.

Loose weights, or parts of weights, render a fork unsuitable for use because a patient may be hearing the chattering sound instead of the tone. For the same reason, forks are not suitable if they have small projecting parts, such as small screws or loosened chips of the nickelled finish, that generate a hissing sound due to the rapid excursions through the air. Likewise, overtones produced by a fork may be mistaken by the patient for the fundamental: with most good forks this factor can be avoided by care in striking the fork at the proper part of the prong. If rubber bands are placed on the prongs of a fork for the purpose of preventing the production of overtones, care must be taken that the bands themselves do not cause a hissing sound: this danger increases as the rubber ages and becomes loosened from the prong.

Where place the stem of the fork? For testing the acuity of hearing by bone conduction the committee recommends that the stem of the fork always be placed on that part of the mastoid region from which the tone seems loudest to the patient when the intensity is well above his threshold. Preliminary tests should be made to locate this area, which may differ for forks of different frequencies: the area found should be recorded for each patient.

Many individuals hear the sound better during bone-conduction tests if the pinna of the ear is touched by the side of the stem of the fork or by the fingers of the hand holding the fork; therefore, if tests by bone conduction are to be comparable, care must be taken not to introduce this variable factor. The pinna should be held out of the way, but in doing so care must be taken that the external auditory meatus is not unintentionally occluded, since this condition would introduce another variable factor.

The same precautions must be observed in using bone-conduction receivers, and for the same reasons.

Calibration of tuning forks. The normal, or reference, standard for any test of hearing acuity must be based on the data obtained from young adults with good hearing. The larger the group of individuals used in establishing the normal threshold for a test, the more reliable the result. However, usually the threshold determined by averaging the tests of a half-dozen young adults with good hearing, selected at random, is very close to the normal that might be found by using a much larger group: and the possible error apt to be thus introduced is less than the other errors due to factors inherent in

all tests of hearing. Certainly threshold standards established by tests of different small groups of young adults form a more reliable basis for the comparison of different groups of data than do standards that consist only of each examiner's own threshold. Therefore the committee recommends that each otologist determine, for his own forks, the "normal air-conduction time" and the "normal bone-conduction time" by averaging the data for a group of young adults. In making these tests, which are to form the "base-lines" for the rest of the calibration, the forks should of course be struck and handled exactly as when patients with impaired hearing are being tested.

When the data obtained from tests made with a fork at one time are to be compared only with the data obtained from similar tests made with the same fork at another time, careful determinations of the normal thresholds, in terms of air- and bone-conduction times, form a sufficient basis for calibration. On the other hand, when the data obtained with one fork are to be compared with those from tests with another fork, or from tests with a bone-conduction receiver, it is necessary also to determine, for both air and bone conduction, the rate of damping and the maximum intensity of sound that can be produced by the fork.

If the rates of damping were the same for all forks, then the maximum intensities produced by each could be calculated directly from the figures for normal thresholds. Unfortunately, this simple relationship does not hold. Forks of the same frequency may have greatly different rates of damping under the same conditions of use; therefore the maximum intensities of sound produced by different forks are not in proportion of the lengths of time that the respective forks can be heard. For example, the maximum intensity produced by a "fifty-seconds" fork may be greater than that by a "ninety-seconds" fork.

Complete calibration of tuning forks is further complicated by the fact that forks with equal maxima for air-conduction tests may differ markedly with respect to the maximum intensities they make available for bone-conduction tests, and vice versa. By direct comparisons on the same groups of patients any two forks of the same frequency can be calibrated in terms of each other, but the data from tests with a large number of different forks can be reliably compared only when the intensity characteristics of the different forks are expressed in terms of some common unit of measurement. The unit that has been most widely adopted for the measurement of the intensity of sounds in general is the decibel.

The decibel is a unit of relative rather than of absolute intensity; and, when used for hearing tests, each intensity is expressed in terms of a geometric progression with respect to the intensity at normal threshold. Since the absolute amount of energy required for normal threshold differs for different frequencies, and since, in the examination of patients with impaired hearing, the primary interest lies in the degree of impairment relative to normal, the decibel system provides a less complicated way of recording the facts of interest to the otologist than does any system of absolute units of energy.

It is a physical property of a vibrating body such as a tuning fork that the reduction in the intensity of the sound emitted is, in decibels, proportional to the time elapsed. Therefore, if the rate of damping of a fork is known in terms of decibels per second, the maximum intensity of sound generated by that fork is, in terms of decibels above normal threshold intensity for the frequency, the arithmetical product of this figure and of the number of seconds required for it to decrease from maximum intensity to that of normal threshold, provided the fork is handled by the same technique in the determination of the rate of damping and of the "threshold time." Likewise, for the same fork and the same technique, the degree of impairment of any given patient is, in terms of decibels, the product of the number of seconds that the patient's threshold time falls short of the normal and the rate of damping per second.

Written out, these procedures may sound very complicated: in practice, however, they are as simple as determining the sales prices of pieces of cloth when the lengths and the costs per yard are known. In fact, for each fork a table can easily be prepared that gives, at a glance, the impairment in decibels that corresponds to each length of time that the fork can be heard.

If the maximum intensity of sound that a fork produces, in terms of decibels above normal threshold, is determined by any method, such as direct matching with the sound of the same frequency as generated by a well calibrated audiometer, then the rate of damping, in terms of decibels per second, may be obtained for different techniques of handling the fork, by division of the figure representing decibels of maximum intensity by the number of seconds that it takes the fork to reach normal threshold when handled by the respective techniques. Once determined, the rate of damping can be utilized to determine degrees of impairment for those patients tested by the different techniques.

During the past decade or so there has been considerable agitation in favor of having all tuning forks calibrated by the U. S. Bureau of Standards, and several otologists are now using forks that have been calibrated at the Bureau. While heartily approving of the general idea, the committee wishes to emphasize a fact that has been pointed out by the staff of the Bureau itself; namely, the rate of damping determined by use of the special apparatus at the Bureau, and called the decrement of the fork, is the correct figure only for the method by which the fork is handled at the Bureau. This method is practically the same as when the fork is used by an otologist for the determination of air-conduction time only, but the method differs in essentials from any technique that can be used for the testing of bone conduction. The rate of damping, or decrement, is useful in enabling an otologist to calculate the maximum intensity of sound produced by a fork, in terms of decibels above normal threshold: and this maximum figure, in turn, is useful as the starting point for the determination of the rates of damping, or decrements, that are correct for the fork when used for the making of different tests.

SPECIAL POINTS ABOUT THE USE OF AUDIOMETERS WITH BONE-CONDUCTION RECEIVERS

Several makes and models of electric audiometers are in use or on the market, and there are several makes and types of bone-conduction receivers, any one of which may be used with any of the audiometers. Some of the audiometers are not calibrated in decibels: the ones most commonly used in this country are, the ones most commonly used in Europe are not.*

For the making of bone-conduction tests, as for air-conduction tests, all audiometers have some very definite advantages over tuning forks. In the first place, measurements of threshold acuity can be made more quickly with an audiometer than with tuning forks, because any given intensity of sound can be maintained until an objective decision is reached as to "heard" or "not heard," and the examiner can more quickly return to any given intensity to check questionable answers. Therefore, in the short period of time that it is practicable to devote to this part of the examination of a patient, the thresholds for more frequencies can be tested with an audiometer than with tuning forks. Secondly, most audiometers and at least

*Two of the members of your committee are now serving on a committee of The American Standards Association occupied with more exact standards for audiometers. These standards, when accepted, should aid in making all audiometers more accurate and the data from the use of different audiometers more easily comparable.

some bone-conduction receivers make available a greater range of both frequency and intensity than do tuning forks.

On the other hand, most of the objections to audiometers for use in making air-conduction tests are also valid for their use in testing the acuity of hearing by bone conduction: an audiometer is an expensive piece of equipment; it is impossible to get repairs or adjustments made quickly in case of accident; any individual instrument may be badly off of correct calibration for one or more frequencies or intensities without the user being aware of the fact; and there are more accidents that can happen to audiometers than to tuning forks.

At the time that this report is being written there are still certain additional drawbacks to the use of audiometers for bone-conduction tests which do not apply to their use for examining hearing by air conduction. In the first place, there is no assurance that any of the bone-conduction receivers now available will hold calibration for a period of years. Secondly, different bone-conduction receivers of the same make and model generate markedly different amounts of acoustic energy for the same flow of current. Therefore, just as with tuning forks, each otologist who uses a bone-conduction receiver must check its calibration at frequent intervals: otherwise the data obtained at one time cannot be accurately compared with those obtained at another time or by another otologist with another bone-conduction receiver. All calibration tests should, as with tuning forks, be made on young adults with good hearing.

The third drawback to the use of bone-conduction receivers at the present time is that we do not have enough correlated information based on data obtained by their use. Therefore it is not yet possible to interpret observations made with bone-conduction receivers as definitely as we *think* we can interpret some of the data from good tests with tuning forks.

It is entirely possible that within a few years the present drawbacks to the use of bone-conduction receivers will no longer exist. The committee believes that manufacturers will succeed in overcoming the present mechanical obstacles and that eventually there will be accumulated enough correlated clinical and laboratory experience to provide a firm basis for the interpretation of bone-conduction tests made with audiometers. The committee also believes that when, in the opinion of conservative otologists, the present drawbacks have been overcome, all otologists will use audiometers for both air- and bone-conduction tests of hearing. Not until this condition is a reality,

instead of a dream, will it be possible to have a real uniformity of method and a truly common language for bone-conduction tests. Then, by using an audiometer in a quiet room, with suitable masking, it will be practicable for all otologists to make simple, accurate and uniform tests of both air- and bone-conduction acuity of hearing, and to communicate their observations to each other with a reasonable certainty that they will be understood.

SUMMARY

The general form of the committee's report has been determined by the fact that it is presented as a part of the Symposium on Hearing by Bone Conduction. Theories, new observations and the interpretation of data fall into the province of the contributions by individual investigators. The committee has attempted to present, in a more or less logical sequence, statements of the basic facts about conditions which affect the accuracy of clinical tests of hearing by bone conduction and the understanding of publications on the subject.

For the most part, the facts presented in the report are not new; and many otologists know of the errors caused by failure to recognize these facts. Nevertheless, they neglect to observe the precautions that they know are indicated by the facts. The usual explanation offered is something like this: "I realize that my tests probably are not exact and that my findings may not mean much to others, but I have learned by experience how to interpret the tests I make with my own instruments under the conditions that I use them. Therefore I hate to change my methods."

While this attitude may possibly be satisfactory for the individual otologist and his immediate work, there is no doubt that it is far from satisfactory from the point of view of the advancement of our knowledge of hearing by bone conduction. From the latter point of view, viz., that of advancing knowledge, it is highly desirable that all otologists make their examinations by methods such that their reports, when assembled, can be collectively utilized in the study of the problems of impaired hearing.

It is hoped that the research papers on our program today will arouse an active interest on the part of many otologists in the problems of hearing by bone conduction and that the work of these otologists will be aided by the outline of the *status quo* contained in this report. The committee believes that the methods suggested in the report for the avoidance of errors in testing hearing by bone conduction are practicable for adoption by otologists generally, and that

the observance of the precautions recommended should increase the value of clinical tests of hearing.

The statements of facts and the recommendations made in the report, which was mailed to all members of the Society in advance of the meeting, are presented under six major headings, subdivided as follows:

I. Introduction.

II. Suitable Sources of Sound for Bone-Conduction Tests.
- *a. For threshold tests.*
- *b. For tests above threshold.*

III. The Effect of Room Noise Upon the Accuracy of Tests of Hearing.

IV. The Use of Masking in Clinical Tests of Hearing.
- *a. Why use masking?*
- *b. Which ear should be masked?*
- *c. How can one ear be masked?*
- *d. Methods of producing satisfactory masking sounds.*

V. Special Points About the Use of Tuning Forks.
- *a. Technique of handling fork after it is struck.*
- *b. Activation of tuning forks. Standard blows.*
- *c. Damaged and otherwise unsuitable forks.*
- *d. Where place the stem of the fork?*
- *e. Calibration of tuning forks.*

VI. Special Points About the Use of Audiometers With Bone-Conduction Receivers.

Of the positive recommendations of the committee the most important are:

1. That all tests of hearing acuity be made in quiet surroundings.

2. That tests of the hearing by bone conduction be made for as great a range of frequencies as is possible with the available instruments, always with due regard to the possible sources of errors.

3. That suitable masking sounds be employed *routinely* while making all tests of the acuity of hearing by bone conduction. Practicable methods of producing satisfactory masking sounds are described.

V. ANIMAL EXPERIMENTS

LX

THE NATURE OF BONE CONDUCTION AS SHOWN IN THE ELECTRICAL RESPONSE OF THE COCHLEA

E. G. WEVER, PH.D., AND
CHARLES W. BRAY, PH.D.

PRINCETON, N. J.

In these experiments the fundamental nature of bone conduction has been investigated. The electrical response of the cochlea produced by bone conducted sound is compared with the response produced by air conducted sounds. This comparison is made, first of all, in terms of the functional relations between the magnitude of the cochlear response and the intensity of sounds conducted to the ear by the two methods. A further comparison is made in terms of the relative effects of middle ear lesions.

The experiments were performed on eleven guinea pigs. Under avertin anesthesia the bulla was opened, and an active electrode was placed in contact with the round window. An inactive electrode was sewn into the muscles of the neck. The electrical impulses picked up by these electrodes were conducted through an amplifier to a cathode ray oscillograph.

Air conducted stimuli were produced by an electric system consisting of an oscillator, filters, attenuators and a loud speaker. A tube from the loud speaker conducted the sounds to the ear of the animal. The tones were of a high degree of purity and of known intensity.*

The apparatus for producing bone conducted stimuli was the same as the above except that a Western Electric bone conduction receiver (Type D-80904) was substituted for the loud speaker. One end of a bakelite rod, 3/16 inch in diameter and 11 inches long was fitted to the plunger of the bone conduction receiver, and the other end was bolted firmly to the exposed parietal bone of the animal. The body of the bone conduction receiver was thoroughly shielded to eliminate electromagnetic radiation.

In order to obtain a measure of intensity of the bone conducted stimuli a vibration microphone was so clamped to the bakelite rod

*For further details, including the method of calibration, see E. G. Wever and C. W. Bray: The nature of acoustic response: the relation between sound intensity and the magnitude of responses in the cochlea, J. Exper. Psychol., 19:129-143, 1936.

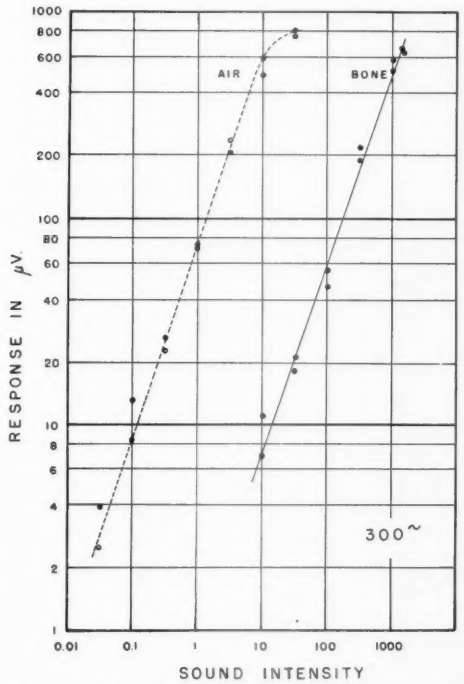

Fig. 1. Intensity functions for air and bone conduction at 300 cycles. For air-conduction curve (dotted) read sound intensity in bars; for bone-conduction curve (solid) read sound intensity in cm. $x10^{-7}$. Filled circles represent one series of measurements, unfilled circles a second series.

as to move with it. This microphone, of the piezo-electric type, produces electrical potentials in proportion to the amplitude of vibration. The potentials, after suitable amplification, were measured with a meter. A calibration of the microphone and associated apparatus in terms of the amplitude of vibration of the rod was obtained by an optical method. A small mirror was mounted on two knife edges, one of which was fixed to the case of the receiver and the other attached to the moving rod. From measurements of the deflection of the beam, and of other dimensions of the system, it was possible to calculate the amplitude of vibration of the rod. By this procedure the intensity of the bone conducted stimuli was found, in terms of maximum amplitude in cm., for the frequencies of 300, 1000 and 3000 cycles. These stimuli were less pure than those delivered by air conduction, especially at high intensities, due to distortion in the bone conduction receiver.

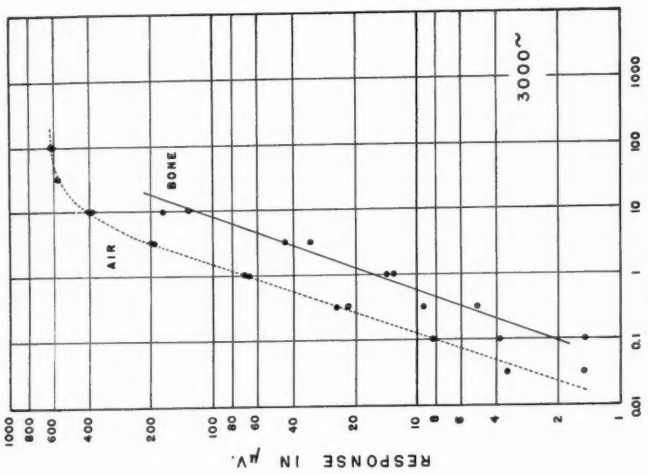

Fig. 3. Intensity functions for air and bone conduction at 3000 cycles. Units of sound intensity as in Fig. 1.

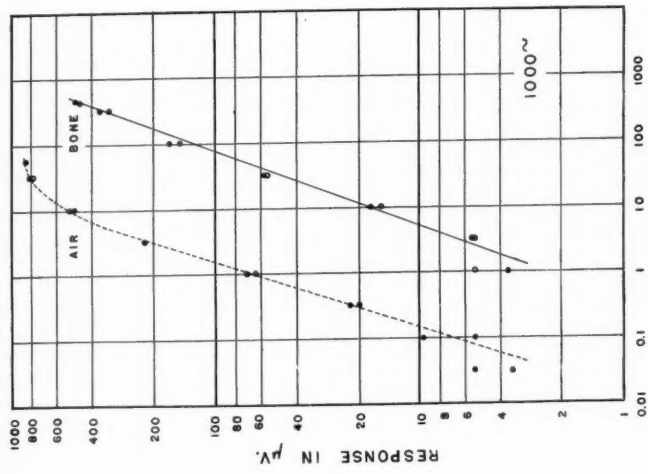

Fig. 2. Intensity functions for air and bone conduction at 1000 cycles. Units of sound intensity as in Fig. 1.

The procedure for each animal consisted of the alternate use of air conduction and bone conduction for the three tones, 300, 1,000 and 3000 cycles. For each of these tones a number of intensities were used, from the weakest that elicited a measurable response to the strongest available. By this procedure normal intensity functions were obtained for the two types of stimulation; a complete series thus included determinations of six intensity functions. At least two such series of measurements were made on each animal, with the ear in normal condition. Additional series were carried out after operative lesions, as described below.

Typical results of the above studies are shown in Figs. 1-3 (animal BC 7, ear normal). Each figure shows intensity functions for air and for bone conduction with a given frequency of stimulation. The magnitude of the cochlear response is shown in microvolts (μv.) on the ordinate, and the intensity of stimulation is shown on the abscissa. Both the ordinate and the abscissa scales are logarithmic. The units of the abscissa scale are different for the two forms of stimulation. For air conduction the scale is to be read as sound pressure in bars (rms dynes per sq. cm.), while for bone conduction it is to be read as maximum amplitude of vibration in terms of ten-millionths of a cm. Since these units are different, and there is no physical way of relating them in terms of their effectiveness in stimulation, no significance should be attached to the lateral separation of the two curves; the curves are placed on the same graph in order to compare their general forms, and to determine the equivalence of the two types of stimulation in terms of response.

For each intensity of stimulus two determinations of response are shown; these were obtained in different series of measurements as described above, and indicate by their degree of agreement the reliability of the method.

It is evident that the functional relationship between cochlear response and stimulating intensity is closely similar for both air and bone conduction. The slopes of the curves are nearly, though not exactly, the same.

It is difficult to make a comparison between the forms of the curves at high intensities. The air conduction curves show a characteristic "bending over" at extreme intensities. In some cases the bone conduction curves also show a slight bending, but the cause is uncertain due to the impurity of the stimuli at the high levels.

In several of the animals the incudo-stapedial articulation was broken, and additional destruction of ossicles and drum was carried

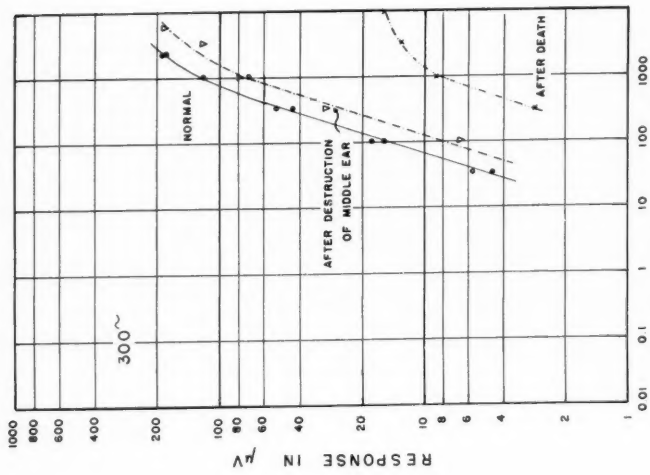

Fig. 5. Responses for bone conduction at 300 cycles.

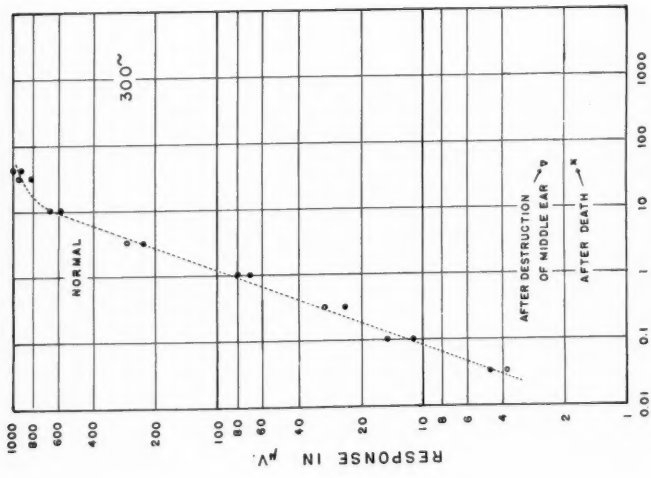

Fig. 4. Responses for air conduction at 300 cycles.

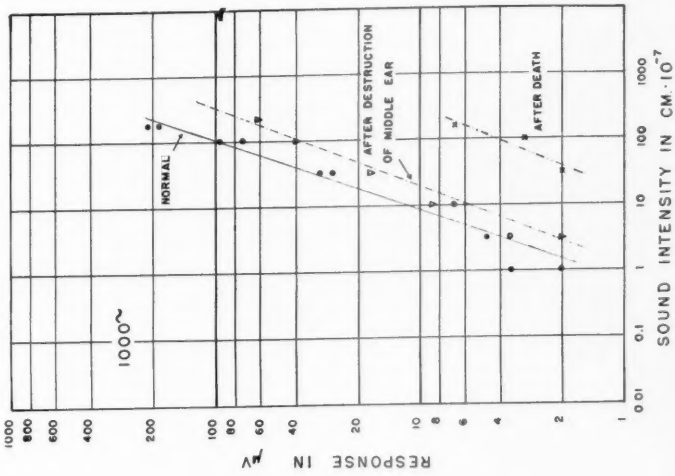

Fig. 7. Responses for bone conduction at 1000 cycles.

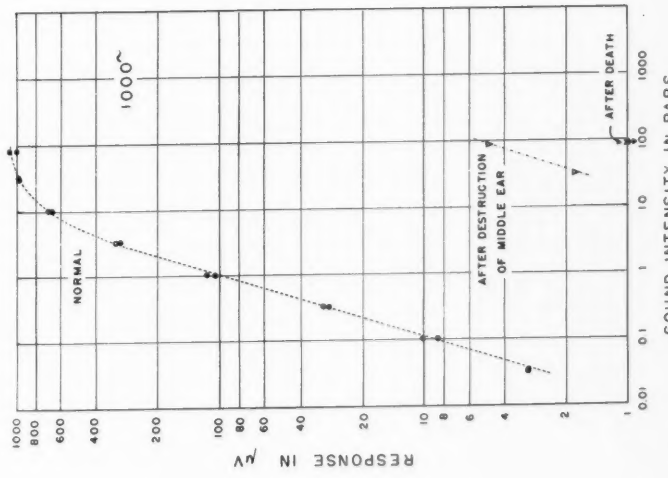

Fig. 6. Responses for air conduction at 1000 cycles.

out when necessary to prevent any re-establishment of contact between incus and stapes. The stapes remained attached to the oval window. After this operation the series of measurements described above was repeated to determine the parts played by the middle ear system in air and bone conduction.

Immediately following these determinations, the animal was killed by pithing the medulla. In this operation, care was taken to remove the blood supply of the cochlea by destruction of the eighth nerve with its blood vessels. A final series of measurements by air and by bone conduction was then made.

Typical results (animal BC 11) for the last two series of measurements are shown in Figs 4-9. Fig. 4 shows results for 300 cycles by air conduction under normal conditions, after destruction of the incudo-stapedial articulation, and after the death of the animal. Only one point was obtained, at a very high intensity of stimulation, for the last two conditions. The impairment of air conduction was very great after disarticulation of the ossicles. Fig. 5 shows the observations for the same tone by bone conduction. After the ossicular disarticulation there was a slight drop of response, and after death a further, considerable drop. Evidently the bone conducted stimulus was less affected by the lesion in the middle ear than was the air conducted stimulus. The results are closely similar for 1000 cycles (Figs. 6 and 7) and for 3000 cycles (Figs. 8 and 9).

In all of the animals studied the responses for air conduction after destruction of the incudo-stapedial joint showed decreases which ranged from 50 to over 80 decibels, with most cases between 60 and 70 decibels. The responses for bone conduction, on the other hand, showed in about half the cases no significant alteration, and in the remaining cases decreases up to 20 decibels. In a given animal, as in the one shown here, the degree of loss was often somewhat different for the three frequencies studied, but in the entire group there was no certain tendency for one frequency to be affected more than any other.

In all cases, the death of the animal and elimination of the blood supply of the cochlea was followed by a rapid decline of such responses as remained.

CONCLUSIONS

The results show in quantitative terms the parts played by the middle ear in the processes of air and of bone conduction. Destruction of the ossicular chain produces a serious impairment of sensitivity

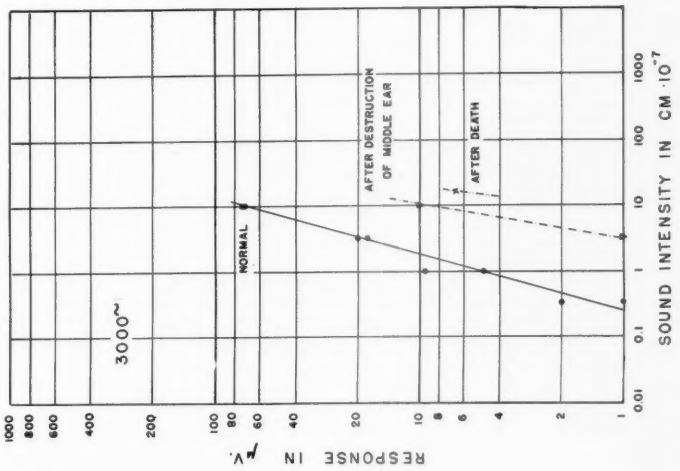

Fig. 9. Responses for bone conduction at 3000 cycles.

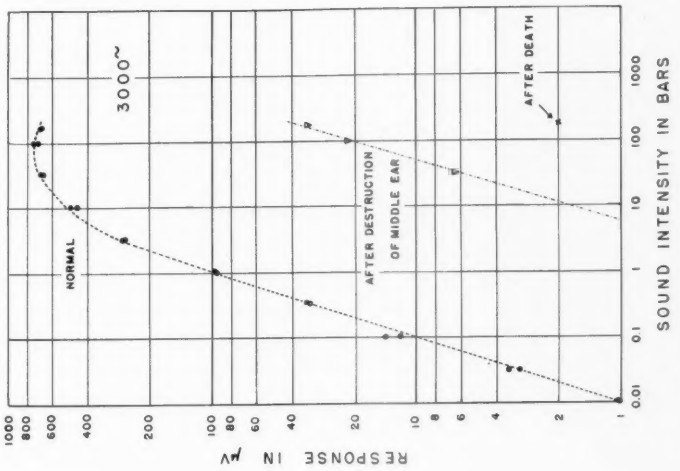

Fig. 8. Responses for air conduction at 3000 cycles.

to air conducted sounds, but has comparatively little effect upon bone conducted sounds. The results for normal conditions show a fundamental similarity in the intensity functions for the two types of stimulation. It seems reasonable to conclude that air- and bone-conducted stimuli involve ultimately the same receptors in the inner ear.

LXI

UTILIZATION OF THE TENSOR TYMPANI MUSCLE REFLEX IN THE ANALYSIS OF THE FUNCTION OF THE MIDDLE AND INTERNAL EAR STRUCTURES

HEINRICH KOBRAK, M.D.*

CHICAGO

The great German physiologist, Carl Ludwig, once expressed his opinion on the progress of our knowledge in physiology with the dictum: "Die Methode ist alles." In recognition of the validity of this statement a considerable part of this paper will be devoted to the demonstration of the experimental method.

In 1879, Hensen discovered that the tensor tympani muscle in a dog moves in response to stimulation of the animal's ear with sound. These reflex contractions of the tensor tympani muscle were studied later by Rockendahl, Pollak and others, and were again thoroughly tested by Kato in 1913. The use of the reflex as a method to test cochlear function in the anmial has been developed only recently. In this country Lorente de No has published important observations using the reflex method in the rabbit.

The contractions of the middle ear muscles, as produced by acoustic stimuli, are simple reflex movements. The N. acusticus is the centripetal part of the reflex. Destruction of the cochlea or cutting of the eighth nerve abolishes the reflex immediately. The central part of the reflex has been experimentally located in the pons. The centrifugal branches are the N. trigeminus for the tensor tympani mus-

*I wish to acknowledge with thanks that the main part of this work has been carried out in collaboration with Dr. J. R. Lindsay and Dr. H. B. Perlman. I also wish to thank the Sonotone Company for furnishing one of their instruments for use in the animal experiments.

cle and the facial nerve for the stapedius. Deep narcosis or death immediately abolishes the reflex. Either labyrinth may elicit the reflex.

The development of modern optical recording technic and the introduction of the electrical sound producer have made possible accurate quantitative measurements of the ratio between tone intensity and strength of contraction.

The slides demonstrate that tetanic contractions of both muscles are produced by a tone, and that the duration of the contraction depends on the length of the sound. When the camera opens there is always a certain click. Since the threshold of the stapedius and the tensor are different, only the more sensitive stapedius reacts to this click. Since the click sound of the camera is always the same intensity and the reaction of the stapedius is equally constant, this furnishes a further proof that equal acoustic stimulations produce equal amounts of contraction.

Any increase of the intensity of the tone is followed by a parallel increase of the muscle contraction. The threshold of the muscle contraction is higher than the threshold of hearing. However, the threshold is not as high as some authors believe, provided certain mistakes are avoided. The fact that the click of the camera elicits a reflex movement speaks for itself.

So far as the mechanical function of the contractions are concerned, several theories have been advanced. The majority of investigators favor the protection theory. However, the accommodation theory and other opinions still have active defenders. The mechanism of the contractions apparently consists in the diminution of the amplitude of the vibrations of the ossicular chain and of the labyrinthine fluid. This effect is, so far as the tensor is concerned, very conspicuous and was demonstrated several decades ago. The errors which the lever method of the early investigators necessarily entail can be avoided by using optical recording. For the routine experiment it is easier not to record the contractions but simply to observe them. The method is very simple. The bulla is opened on its ventral side. A 15-diopter lens provides a suitable enlargement of the tensor. The observer determines the intensity of a tone which gives a threshold contraction of the tensor. The readings are taken for different pitches and the results plotted like audiometer curves. The threshold contractions of the tensor are very definite and various observers do not differ more in their readings than do audiometer tests in the clinic.

The rabbit is kept in superficial general anesthesia. Dealing with a reflex, the question of anesthesia is a very important one. The curve taken on an animal under local anesthesia, compared with the average threshold reading for sixty normal rabbits under urethane anesthesia demonstrates that with this type of narcosis there is hardly any interference with the reflex movements. The tensor contractions may be observed continuously over a period of several days provided aseptic precautions are taken.

One of the greatest assets of the optical method is the fact that the reflex can be observed in the human. The contractions of the stapedius muscle in the human were first seen by Lüscher, who observed the tendon with his special otoscope. The number of patients who have a perforation in the posterior half of the drum that permits observation of the stapes or the stapedius tendon is not small: we have seen about thirty cases during the last year in the University of Chicago clinics. In some of these patients the movements of the stapes was recorded by means of a moving picture film (Demonstration).

The reflex works in the same way for bone conduction. As a sound source the audiometer connected with a bone conduction receiver has been used. In the animal experiment the receiver was placed on the teeth, in the human usually over the mastoid process.

The value of the reflex contractions of the middle ear muscles, as an objective means to determine cochlear function, may be summarized briefly as follows:

1. In the rabbit the two muscles of the middle ear contract regularly in the presence of a tone of a certain intensity and frequency. The contractions begin and end accurately with the tone.

2. The reflex is elicited by both air and bone conducted sounds.

3. The ratio between the intensity of the sound and the strength of contraction is definite. Increase or decrease of intensity is followed by parallel changes in the muscle contraction.

4. The threshold for the muscle contractions, determined by observation with a lens, is higher than that for hearing.

5. Low and high tones are less effective than the middle range frequencies in producing these reflex contractions.

6. In man reflex contractions of the stapedius muscle can be observed, which show nearly identical properties as found in the ani-

mal. Numerous comparisons between reflex activity and hearing sensations can be made.

EXPERIMENTS ON THE RINNE, WEBER AND SCHWABACH TESTS

The following lesions of the sound conduction apparatus have been produced in the experimental animal: (1) Plugging of the outer acoustic canal representing the conditions in man with cerumen obturans. (2) Filling the bulla with fluid, thus simulating middle ear catarrh. (3) Partial destruction of the ossicular chain; for instance, perforation of the eardrum, destruction of the incudostapedial junction, luxation or extirpation of the incus, thus producing conditions comparable to those in some patients with chronic suppurative middle ear lesions. Some of these procedures have the advantage of being reversible, in that a record can be taken not only before the lesion has been produced, but rechecked after removal of the lesion. Its importance is evident if only slight changes are expected. The ability to repeat the observations assures results which otherwise would not be reliable.

In the study of the Rinne test in the animal the threshold for contraction of the middle ear muscles for air conduction was taken first and then the threshold for bone conduction. Since the sound producing apparatus for air conduction and bone conduction is not the same, we cannot compare the two thresholds from the quantitative standpoint. But this is also true in the clinical Rinne test, where the prongs and the stem of the tuning fork are different sound producers with different physical qualities. Having determined the normal thresholds, if one produces lesions of the sound conduction system, the hearing for air conduction goes down or even disappears entirely. At the same time the hearing for bone conduction does not show any impairment, but is even improved to some extent. Our experiments show that these functional changes take place regardless of the nature of the lesion to the conductive apparatus. The plug in the outer acoustic meatus has the same influence as the destruction of the ossicles or the instillation of fluid into the middle ear. However, the degree of change may be different; in some instances, the air conduction is absolutely lost for intensities produced by our apparatus. Plugging of the outer canal produces only a minor impairment. These experiments illustrate that the conditions of the Rinne test may be observed in the animal. Lesions of the sound conducting apparatus create conditions which alter the normal proportion between air and bone conduction so that finally the bone conduction may be better than the air conduction. Lesions of the inner ear

diminish or abolish the function for air and bone conducted sounds alike.

The Weber test cannot be carried out experimentally in the animal for the following reasons: the middle ear muscle reflex in either ear is produced almost equally by either labyrinth. Besides this physiological reason there is a psychological reason, which involves the very nature of the Weber test. The fact that a patient lateralizes to one ear does not mean that he has no acoustic irritation in the other ear. The sensation is shifted into the one ear, due to a psychologic effect, while the appreciation of the acoustic stimulus in the other ear is suppressed. Under these conditions hearing sensation and acoustic irritation are dissociated, and the middle ear muscle reflex fails to be a true picture of the hearing sensation.

The experimental procedure for the Schwabach test in the animal is similar to that used for the Rinne test. The readings of the tensor are made before and after the lesion has been produced.

In this country evidence has been presented which seems to indicate that an absolute increase in bone conduction does not exist. It has been assumed that in making tests on bone conduction in a room which is not soundproof, the noise of the environment has a marked influence on the results. Our experiments show that the threshold for bone conducted sound is slightly but definitely lowered after lesions in the middle ear. For practical purposes it may be sufficient to do clinical tests in a soundproof room and to omit any discussion whether or not there is an additional real increase in bone conduction which is not explainable by the masking effect. If, however, we wish to understand the problem of bone conduction, we cannot discard this experimental finding, even if the difference is minute from the quantitative standpoint. The animal experiment in which the cochlear function can be measured reliably seems to furnish a new way of approach. Since we observed a stronger reflex contraction after the middle ear lesion had been made, the most obvious explanation is that the irritation in the inner ear was slightly greater than before.

EXPERIMENTS ON MASKING

In this series of experiments the influence of a masking tone on the contractions of the tensor tympani muscle produced by bone conducted sounds has been studied. The work has been done in collaboration with Dr. Elisabeth Huff.

Both pure tones and noise were used as masking sounds. We found two distinctly different results according to the intensity of the masking tone. If the masking tone is above the tensor threshold, the tensor is in a stage of contraction so long as the masking tone lasts, and the primary tone produces superimposed contractions. In this case the determination of the superimposed contractions cannot be made accurately. Because of this interference with the muscle itself, we discarded loud masking tones and confined the intensity of the masking tones to under-threshold values.

If the masking tone is below the tensor threshold there is no appreciable change in the contractions due to the primary tone. Observations were made on human beings, employing the same frequencies and intensities as were used in the animal experiments. As was to be expected, a typical masking effect was obtained. A tone that is distinctly effective for masking in the human does not interfere with the tensor tympani reflex. The conclusion may be drawn, therefore, that the centripetal impulse originating in the cochlea and determining the strength of the tensor contraction is the same, regardless of the presence or absence of a masking tone. The stapedius reflex observed in man corroborates the findings of the animal experiment. There was no noticeable change in the stapedius contractions due to a bone conducted sound when a masking tone was introduced. Needless to say that the hearing sensation showed a typical masking effect when tested with the same masking tone. Incidentally electrical experiments by Davis and Saul gave a similar result. The cochlear potentials picked up at the round window did not show interference by masking. The phenomenon of masking apparently is not located in the peripheral sense organ.

In addition to these experiments, some more recent observations may be described; although this work cannot be called completed yet, it may be mentioned here in the form of a preliminary report.

FUNCTIONAL CHANGES DUE TO LOSS OF LABYRINTHINE FLUID

If a very fine hole is drilled in the apical part of the cochlea of the rabbit an increasing functional impairment is observed as the labyrinthine fluid escapes. The effect is about equal for bone and air conduction. If the drill-hole is allowed to heal, one can observe some remaining function, since the inner ear apparently is now under more favorable hydrostatic conditions. Briefly this series of experiments suggests that the labyrinthine fluid is an essential part for the cochlear function and is equally important for air and bone conducted sounds.

FUNCTIONAL CHANGES DUE TO EXTIRPATION OF THE STAPES

Experiments have been carried out in which the stapes was destroyed or extirpated in the rabbit and the functional changes for bone conducted sounds observed. If one destroys the crura of the stapes there is no loss of bone conduction. The destruction of the stapes footplate of course opens the labyrinthine fluid system. A gush of fluid escaping from the oval window indicates the successful destruction of the stapes footplate. Now a piece of cotton must be inserted immediately. With this method it is possible to prevent a too severe loss of labyrinthine fluid. The result has been that cochlear function can be demonstrated for bone conducted sounds, even in the absence of the stapes.

FUNCTIONAL TESTS AFTER PLUGGING OF BOTH WINDOWS

Both the oval and round window were plugged with plaster of Paris and the resulting cochlear function determined. No cochlear function could be demonstrated, by either air or bone conducted sounds, after such blocking.

DISCUSSION

The three series of experiments were carried out with the purpose of obtaining information regarding the pathways of bone conducted sounds on their way to the organ of Corti.

The experiments on the middle ear indicate that good bone conduction is compatible with complete absence of the ossicular chain (drum, malleus, incus and the cura of the stapes). The additional destruction of the stapes footplate causes a definite drop in bone conduction. The result is explainable by the loss of labyrinthine fluid. It was experimentally impossible to determine the isolated effect of the stapes footplate on bone conduction. Bezold's opinion that the bone conducted sound can enter the labyrinth only via the stapes has not been corroborated by these experiments. Apparently cochlear irritation by bone conducted sounds is possible in the absence of the complete ossicular chain.

The escape of intralabyrinthine fluid is associated with a severe loss of cochlear function. Intralabyrinthine fluid is vital for cochlear function as tested by either air or bone conducted sounds.

A possible explanation for the absence of cochlear function when both windows were solidly fixed by plaster of Paris is that the aqueductus cochleæ and the related lymphatic craniolabyrinthine commu-

nications possess a too great resistance to act as a third window and permit displacement of fluid. The experiments therefore seem to corroborate the teaching of Rejto and Herzog, that bone conducted sound produces rhythmical vibrationts of the otic capsule. These vibrations irritate the end organ identically as it is irritated by air conducted sounds.

LXII

THE CONDITIONED-REFLEX METHOD IN THE STUDY OF HEARING BY BONE CONDUCTION: THE DIFFERENTIATION OF TACTILE FROM AUDITORY RESPONSES

E. A. Culler, Ph.D.

Urbana

What I have to say will be concerned largely with certain procedures and apparatus which prove to be useful in the "conditioned response" method of investigating hearing by bone conduction.

Two bone conduction receivers were used: a Western Electric model and an acousticon, which were kindly donated to us by the manufacturers.

To investigate the frequency response of these two receivers we utilized the type of instrument mentioned by Dr. Wever. That is to say, if a quartz crystal bar, properly cut, is firmly mounted at one end and a receiver attached to the other, the electrical potentials generated in the bar itself may be measured by the mechanical stresses impressed upon it from the attached receiver. The quartz bar is silvered on each side, and the potentials generated are led off through an amplifier and accurately evaluated. This is a standard procedure for gauging the relative efficiency of mechanical oscillators at various frequencies. The two are compared in table 1.

The figures in rows 2 and 3 show for each frequency the relative amount of electrical energy (in decibels) needed to give a certain

Table I

Frequency	125	250	500	1000	2000	4000	8000
W. E.	—14.7	—22.8	—25.2	—34.0	—30.3	—29.3	— 6.5
Acousticon	— 6.3	—23.3	—33.8	—43.3	—35.7	— 6.3	+ 4.0
Difference	— 8.4	0.5	8.6	9.3	5.4	—23.0	—10.5

constant mechanical energy in the receiver. Thus at 125 cycles, the W. E. receiver required 8.4 db. less electric energy than did the acousticon to give the same mechanical response; the W. E. is obviously more efficient at this frequency. At 1000 cycles, however, the W. E. requires 9.3 db. more electrical energy and is therefore less effective in converting electrical into mechanical energy than is the acousticon at this frequency. In general, the acousticon is less efficient at the lower and the higher frequencies and more efficient at the intermediate frequencies. The W. E. is more nearly uniform in its frequency characteristics than is the acousticon. Both receivers, I may add, were tested under optimal conditions, so that our figures represent the best performance of which they are capable.

The next problem was: how shall the receiver be attached to the animal? Obviously, in quantitative work, one cannot use an ordinary headband or something which merely presses the receiver against the skin at some point of the head. The contact would vary so much as to make accurate measurements impossible. The procedure adopted was to anesthetize the animal and incise the skin along the zygomatic arch; a silver screw is then turned directly into the bone. When the receiver is attached to the screw, the animal is free to move the head about and evinces no discomfort of any kind.

Next we need to consider procedure or methodology. The general method of "conditioning" animals is familiar, I think, to all of you, on the basis of general information and previous reports to this Society; hence I shall be very brief here.

The animal is first trained to react to an air-borne sound by withdrawing the paw from a charged electric grid; every time he hears the tone he flexes the leg and thereby escapes punishment. As soon as the dog is well trained we destroy one cochlea and incise the skin for attaching the bone conduction receiver. The transfer from air to bone conduction is extremely simple. If the animal is trained to respond to a sound by air, he will soon respond equally well to the same tone when administered through the bone conduction receiver.

The critical question that must be considered here has to do with tactile sensation and the rôle which it may play in these tests. In other words, the question arises: If the animal responds, are we certain he is responding because he hears a sound or simply because he feels, by tactile sensibility, the tremor of the receiver?

I do not know any convenient method of destroying or obviating tactile sensibility in the head; but fortunately we can, for

practical purposes, meet the difficulty in this way. Table 2 exhibits the normal thresholds for both bone and air conduction at 125, 1000 and 8000 cycles.

TABLE 2

	Bone			Air		
Frequency	125	1000	8000	125	1000	8000
Cochlear	—57	—75	—51	—73	—98	—74
Noncochlear	—19	—22	0	—12	0	0

These figures signify that the dog's threshold was 73 decibels below our standard intensity at 125 cycles, 98 db. below the standard level at 1000 cycles, and so on. These are called "cochlear" thresholds because one cochlea is still intact and functioning. The remaining cochlea is then destroyed and the "non-cochlear" response determined. We now find that the dog does not react to 8000 cycles at zero, the strongest sound we have. At 1000 cycles also he fails to respond at zero; so his present threshold is obviously higher than our loudest standard sound. At 125, however, the dog responds down to —12 db., whereupon all reaction ceases.

Several interpretations of the "noncochlear" thresholds are conceivable. We might say that hearing continues in complete absence of a cochlea. Much more likely is the assumption that the animal now responds to tremor instead of to sound. I should therefore call the noncochlear response a tremor threshold. At 125 cycles our large dynamic speaker, well baffled, communicates to the walls and furniture of the test room a tremor which is readily perceptible. The animal must also perceive this tremor effect. In brief, we have here a wide range (12 to 73, and 0 to 98, and 0 to 73 decibels) within which tremor is not a factor and only the cochlear response participates. Anything within these limits may safely be called a true acoustic or cochlear response.

In the case of bone conduction, the situation is similar. At 8000 cycles the dog does not respond to our greatest intensity. At 1000 cycles he responds down to —22 db. and at 125 down to —19 db. We therefore conclude that down to —19 and —22 db. we are dealing with a noncochlear or tremor threshold; below that, down to —57, —75 and —51 decibels, we have true hearing.

While we cannot readily abolish tremor sensibility in the animal, we have in practice a wide field below that level where the responses are due to acoustic sensitivity alone.

As for actual observations, I have only one or two of a preliminary and exploratory kind. A few experiments were made on the hearing loss sustained after lesions of the tympanic membrane. A small hole, about one square mm. in area, directly back of the malleus (posterosuperior quadrant) had no effect whatever on bone conducted sound. The thresholds at all three frequencies before and after the lesion were identical; whereas with air borne sound a loss of 12 db. at 125, 18 db. at 1000 and 18 db. at 8000 occurred. Two small holes, one directly behind the malleus (posterosuperior quadrant) and one directly in front of it (anterosuperior quadrant) likewise had no appreciable effect on bone conducted sound; but the air borne sound registered a loss of 15, 20 and 21 db—in each case slightly more than the reduction due to a single perforation.

By the methods above described, we can differentiate accurately between the cochlear and the noncochlear responses, and thereby eliminate tactile sensibility as a factor in the study of bone conduction hearing in dogs.

LXIII

TACTILE AND TREMOR PERCEPTION

S. DWORKIN, M.D.
G. A. HUTCHISON, M.D.
AND
J. KATZMAN, M.D.

MONTREAL

For some time we have observed that cats, rendered by operation totally deaf, can still perceive very intense sounds. The object of the present research is to determine the receptors involved in picking up these powerful sound stimuli. Upon the subject immediately under discussion our results may have only an indirect bearing except in so far as responses to bone conduction tests in animals must often be attributed to tactile stimulation.

The subject was studied in cats, by the motor-alimentary conditioning method already often described from our laboratory. The normal hearing acuity was first established for frequencies between 100 and 16,000 cycles per second. Then each cochlea was pierced

with a drill one-half millimeter in diameter. This operation, which causes no visible cochlear bleeding, makes the animals completely deaf. As shown by a control animal, in which both cochleæ were completely destroyed, the small drill hole is just as effective in abolishing hearing as is the more elaborate operative interference.

In each totally deaf animal responses to the emission of powerful sounds (at frequencies from 40 to 1,000 cycles per second) were registered. In one case the upper limit was about 1,200 cycles. For frequencies between 100 and 1,000 cycles the thresholds were determined as carefully as possible. The sounds were measured indirectly in three different ways: (1) by reading the current in the loud speaker; (2) by using a calibrated attenuation meter across the loud speaker; (3) at some frequencies, by comparison with the tone from a standard audiometer. As a reference standard we used the hearing thresholds of cat and man at 1,000 cycles (cf. Fletcher, 1933). In all tests the head of the subject (whether cat or man) was directly in front of the loud speaker, twenty inches distant.

Audiograms for these totally deaf animals are shown in Fig. 1. The sharp cut-off at frequencies about 1,000 cycles is characteristic. The threshold intensities, as nearly as we can measure them by indirect methods, vary from 100 decibels above normal hearing at 100 cycles to 75 decibels at 900 cycles, while at 1,000 cycles the threshold note was decidedly painful, which, according to Fletcher's findings, implies an intensity of at least 120 decibels.

The question now arises, what end organs are concerned with reception of these loud tones? The probability is that they are not nerves of ordinary pain. For one thing the threshold intensity was much below the 120 decibel level. Furthermore, no responses were observed at high frequencies, whereas intense tones as high as 16,000 give rise to the sensation of pain. Our apparatus produces a note of an intensity painful to man at frequencies of 2,000 or even 5,000 cycles, but no responses were ever elicited from the deaf cats by these tones.

We then considered the possibility that the loud sounds first of all caused tremors to be set up in the table on which the cats were standing. On this supposition the ultimate stimulus would be of a tactile nature. If this were true, then the thresholds found by us would be really the thresholds at which the table could be set in palpable motion. We tested out this possibility on human subjects. They were required to place their hands on various parts of the table, and

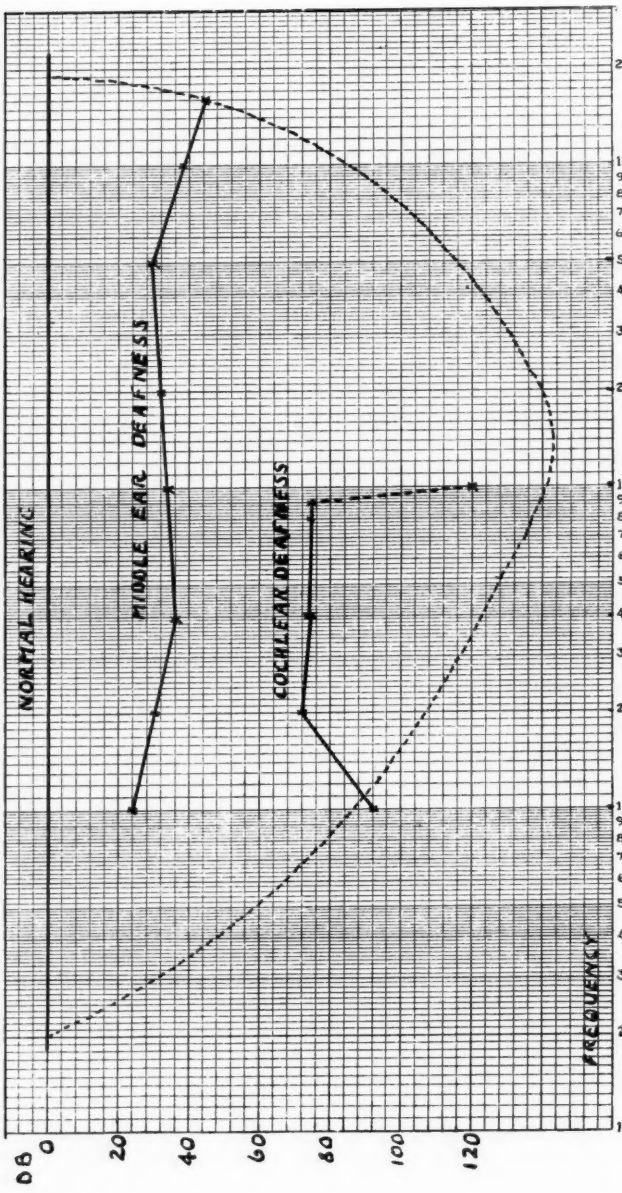

Fig. 1. The curve marked "cochlear deafness" represents thresholds of response to the intense sound fields at frequencies between 100 and 1,000 cycles per second. Note the sharp rise at the latter frequency. This curve can be compared with the thresholds for normal hearing (top line) and those of experimental middle-ear deafness (middle line).

to note the presence or absence of palpable tremors when the cat-threshold sounds were produced. Such tremors were definitely felt at the threshold for frequencies between 100 and 600, but very uncertainly for frequencies above 600 cycles. Thus the correspondence was at least not complete.

Before concluding that the animal's tactile sensibility between 600 and 1,000 is keener than that of man, two other sets of experiments were made. In one we compared the responses of dog and man to a specially built tactile stimulator. This was a modified magnetic speaker unit, driven by our audio-oscillator. By means of this instrument tactile stimulation was applied to the head and body of a dog, and to the head and finger of human subjects. Responses were readily observed in man and animals at frequencies between 60 and 1,500 cycles per second; sometimes at 2,000 cycles. Whether at the higher frequencies 1,500 or 2,000 stimuli per second are transmitted along the sensory nerves is, of course, very difficult to say, in view of our knowledge of nerve conduction. We must bear in mind the results obtained by Allen on tactile stimulation in man, where an upper limit of 500 stimuli per second was observed. What is of interest in our experiments is that both for dog and man the frequency range was the same.

In another series of experiments we decided to stimulate the animals by direct vibrations of the table with very little accompanying sound. These vibrations were produced by means of a strong magnet, driven by the audio-oscillator in a powerful field. The vibrations so produced could be distinctly felt by man at frequencies between 30 and 500 cycles. In the cat, an exactly similar upper limit was observed, which accords well with Allen's observations as to the upper limit for direct tactile stimulation. These conditioning experiments on deaf cats subjected to vibration through the medium of the table on which they stood seem to show fairly clearly that the upper limit of their feet or legs for direct tactile vibration is in the neighborhood of 500 cycles per second.

Reception of the sound frequencies between 500 and 1,000 still stood in need of some form of explanation. Consequently we placed the animals on a medium which would not vibrate at the usual sound-tremor thresholds. In turn we used for this purpose soft rubber, felt, heavy plates of lead, and then a pad composed of all three of these materials. When the cats stood on these substances, the thresholds were not, however, appreciably affected, particularly at the higher frequencies (600 to 1,000 cycles).

A very satisfactory material was finally found; it is a loosely strung fine-wire mesh, supported on small posts of soft rubber. This material proved to be an excellent screen against mechanically conducted tremors. Not only did it obliterate the threshold tremors to one's hand held in contact with the wire surface, but it equally screened against tremor when the sound intensity emitted by the speaker was greatly increased. When the deaf cats were placed on this material and retrained, the responses occurred at the previous thresholds. As we had thus satisfactorily excluded the occurrence of tactile stimulation by way of the feet, at all frequencies up to 1,000, we were forced to conclude that the stimuli were acting on receptors other than those in feet or legs. While the possibility of some extra-cochlear, cranial receptor cannot be entirely excluded, a more obvious source of tremor reception turned out to be the body surface of the animals, particularly the fur! The unexpected but conspicuous ability of a cat's pelt (bearing its fur) to pick up and to hand on tremor at the threshold intensities was tested and definitely proved. If one may argue from the qualities of the dried pelt to the skin of the living animal, it seems reasonable to conclude that in cats the body surface may provide a good medium for amplificatory reception of loud sounds.

The extent of discrimination possible between sound stimuli registered in this way has been tested and proved to be very poor. While the normal cat can easily discriminate between 800 and 600 cycles, deafened cats could distinguish with difficulty only 800 from 60 cycles. Even then the differentiation depended more likely on intensity than on the pitch difference. The practical use of sounds of this sort seems, therefore, to be greatly limited.

LXIV

AN EXPERIMENTAL STUDY OF BONE CONDUCTION*

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The present report represents a series of experiments begun four years ago in the Otological Laboratory of the Johns Hopkins Medical

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School. The earlier experiments were largely confined to the development of a reliable experimental technic whereby the phenomenon of bone conduction could be studied by measuring the changes in electrical potential set up in the auditory nerve by bone conducted stimuli. The problem of bone conduction has been studied extensively both from the clinical and experimental standpoint. In general two problems have been investigated. An effort has been made to establish the pathways by which bone conducted stimuli are brought to the sensory cells of the ear and to determine whether or not bone conduction could be affected by middle ear pathology or experimental procedure. The problem has been so complex that a complete analysis of results obtained is difficult. However, certain observations have been made and their possible significance will be indicated. The experiments detailed below have been carried out in the Otological Research Laboratory of the Abington Memorial Hospital in the service of Dr. George M. Coates. This laboratory, which was recently designed and constructed, is illustrated diagrammatically in Fig. 1 and is arranged to permit of both experimental and clinical studies.

The arrangement of the laboratory and its electrical equipment is as follows: The sound proof chambers consist of two separate rooms, an outer room of concrete block and an inner room of wood construction entirely isolated from the outer room and set on springs. The space between the rooms contains sound attenuating materials and specially constructed sound proof doors are used. Both the sound proof room, which is 14 by 6 by 8 feet inside, and the sound proof booth, which is 4 by 4 by 7 feet inside, are shielded against electrical interference by galvanized sheet iron.

The tone source is a General Radio beat-frequency oscillator with a frequency range from 50 to 20,000 cycles. The output of the oscillator is controlled by a special attenuator with a total attenuation of 100 decibels, variable in five decibel steps. The output of this attenuator is passed to a Western Electric 555W dynamic loud speaker, which serves as the sound source. All of this apparatus is separated from the sound proof, electrically shielded room containing the experimental animal. The sound from the loud speaker is conducted into the sound proof room by two three-quarter inch tubes consisting of ordinary garden hose, branching out from the loud speaker through a siamese connection and passing through the room wall. A plug type valve in each tube line, as close to the siamese as possible, controls the transmission of sound through the tube. If one valve

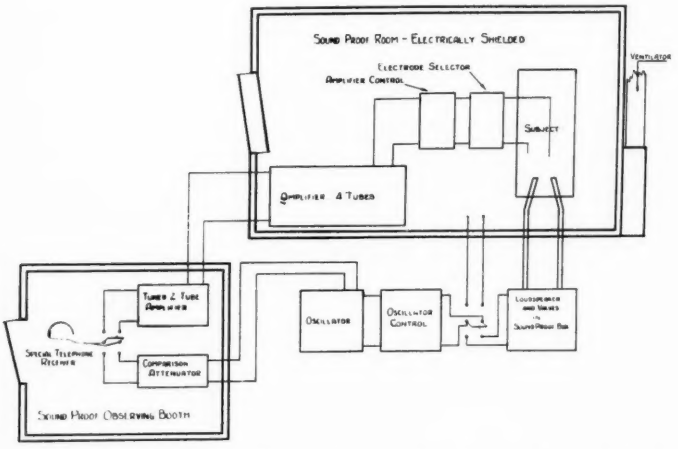


Fig. 1. Diagram of sound proof room and booth, showing position of sound source, amplifier, and balancing system. The ventilator is connected with the hospital ventilating system and is sound proofed by a concrete baffle at both inlet and outlet.

is closed the stimulus is delivered only to the ear being tested. When both valves are closed acoustic and electric pick up can be checked. The hose lines pass through the operating table and terminate in glass tubes which fit into the animal's external ears. The tubes are kept from contact with the table and the pinnas by cushioning with sponge rubber. This is done to prevent bone conduction stimulation by vibration of the side of the tube tranmsitted through the table to the animal.

The electrodes, nerve or cochlear, are connected to a selector switch which enables change from one electrode to another to be made without disturbing the animal. The impulses from the electrodes pass into the amplifier directly and if too intense are passed through an attenuator first and then into the amplifier. This amplifier consists of two units, the first containing four stages of vacuum tubes of a type selected for their property of giving high amplification with very low accompanying noise. These tubes are inherently microphonic and must be protected against mechanical and acoustical vibration and shock. This is done by suspending each tube on springs and enclosing all four in a sound insulated cabinet. Each tube is placed in a case iron box for shielding against magnetic fields, and the cabinet as a whole is shielded by sheet iron. The second unit is housed in a sound proof booth adjacent to the sound proof

room. This unit, containing less sensitive tubes, is constructed on a rack.

The impulses from the electrodes are observed by means of a dynamic head receiver connected to the second unit of the amplifier. Sound impressed on the ear of the animal is heard in the receiver. It is impossible to obtain large amounts of amplification without an accompanying electrical disturbance, heard in a telephone receiver as a hiss, which makes measurement difficult. This difficulty can be overcome by tuning the amplifier to the frequency of the tone being observed. This is done in the present case by tuning the first stage of the second unit.

Changes in intensity of impulse received from the animal and heard in the telephone receiver are measured by comparison with a sound of standard intensity in the following manner. A small fraction of the output voltage of the oscillator is conducted into the sound proof booth, passed through an attenuator, amplified and then observed with a telephone receiver. Changes in attenuation of the oscillator control attenuator (which alters the intensity of sound delivered to the ears of the animal) have no effect on the sound heard in the telephone receiver connected to this system, which is designated "comparison system," its attenuator being called "comparison attenuator." Hence by maintaining constant output voltage at the oscillator the sound at the "comparison system" is always constant for zero setting of the "comparison attenuator." The tone in the "comparison system" is made equal in loudness to the tone from the animal by adjusting the "comparison attenuator." The reading of this attenuator then gives the volume of the sound from the animal in decibels below the standard tone of the same frequency. Changes in readings are then the same as changes in impulses received from the animal.

In all of the experiments measurement of changes in electrical potential of the auditory nerve has been used as the standard of response. The nerve electrode has been used in preference to the cochlear electrode for two reasons. First, the majority of our earlier observations on air conduction were made by this method, and second, it also permits measurements without opening the bulla. It has been definitely established that simply opening the bulla may modify the bone conduction response. Howe, working in the Hopkins laboratory, demonstrated this effect repeatedly and found further that plugging the opening of the bulla with a pledget of dry cotton increased the response of the ear by bone conduction. To

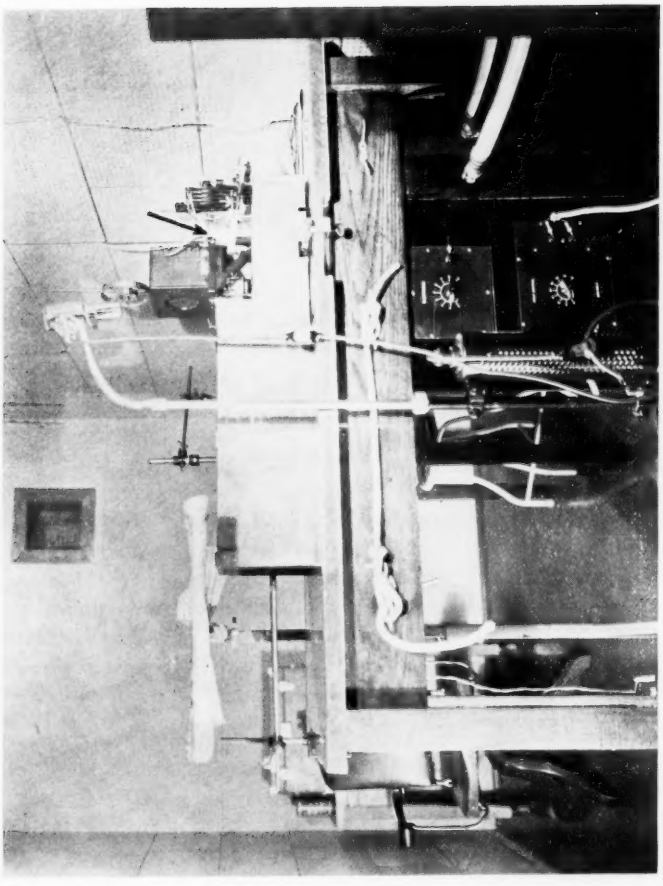


Fig. 2. Setup of bone conduction experiment showing operating table, hose connection from dynamic receiver, electrode switch, amplifier control and copper shield. The arrow indicates bone conduction receiver in position with tip resting upon screw in hard palate.

study this phenomenon adequately, therefore, the use of the nerve electrode is essential and it has been used whether the bulla was opened or not.

A variety of methods have been used to stimulate by bone conduction. Kobrak et al.[1] applies the vibrating mechanism to different parts of the skull. This is, of course, the obvious and natural method, but in experiments of this type it is necessary to move and replace the bone conduction rod frequently. Experience has shown very definitely that these changes in position, no matter how carefully the rod is replaced, may of themselves cause appreciable changes in response. To establish an absolutely constant experimental condition, a metal screw is inserted in the hard palate 0.5 cm. posterior to the incisor teeth and the tip of the receiver rod allowed to rest upon the head of the screw. The bone conduction receiver always rests with the constant pressure of its own weight. This receiver consists of a flat, rectangular piezo-electric crystal (Rochelle salt) mounted in a brass case in such a manner that three of the corners are clamped tightly and held fast by small rubber feet. A bakelite tip is fastened to the fourth corner, which is free to vibrate. In operation the receiver generates an electric field, which must be shielded from the cat and amplifying equipment. This is accomplished by surrounding the animal with copper, the receiver tip passing through the copper shield. Fig. 2 illustrates the operating table as set up for a bone conduction experiment with the copper shields and receiver in place.

In studying bone conduction by changes in electrical potential of the auditory nerve several sources of error must be recognized and guarded against. Acoustic pickup in the experimental animal is as important a factor as it is in similar bone conduction tests in man. This is avoided by using an intensity of stimulus so low that repeated control tests fail to produce any acoustic response. Another troublesome source of error has been the effect of mechanical vibration upon the electrodes. This particular factor develops, regardless of the type of electrode used. Protection can be obtained by removing the bone conduction receiver from the table and fixing the animal's head in such maner that neither room nor receiver vibrations can introduce any extraneous factor. The final test for the presence or absence of this vibration is stimulation some time after death. If the intensity of the stimulus is too great the response is obtained indefinitely post-mortem. It can be seen, therefore, that a stimulus of low intensity must be used, which represents in general an intensity close to the

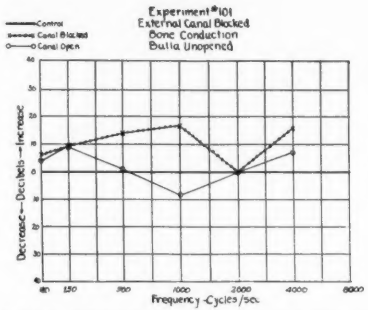
Fig. 3. Removal of the septum impairs B. C. while A. C. is not affected. 8000 not transmitted by B. C. in control test.

Fig. 4. Note slight impairment in B C., instead of improvement, which occurs when the external canal is blocked in an ear with intact bulla. (Compare with figure 5.) A. C. is almost entirely lost but transmission returns to control level when gauze is removed. 8000 not transmitted by B. C. in control test.

animal's threshold for bone conduction. Another complicating factor is the invariable cross effect from one ear to the other. If one ear remains normal this cross effect will definitely enter into the response obtained from the experimental ear. In the majority of experiments, therefore, it is necessary to destroy one ear completely to obtain acurate measurements of changes produced by procedures carried out on the experimental ear. At first the cochlea was destroyed mechanically, but occasionally this was found to produce rather profound shock, so the present routine is to place sodium chloride crystals directly against the round window membrane. All response, both by air and bone conduction, disappears in about thirty minutes following this procedure. An interesting immediate effect of applying the sodium chloride to the round window is a definite improvement in air conduction. A possible explanation of this response is temporary fixation of the round window membrane before the sodium chloride has begun to exert its osmotic effect upon the intralabyrinthine fluid.

Careful elimination of these sources of error, therefore, is necessary before any conclusions may be drawn from the experimental data obtained. The animal (cats have been used exclusively) is set up in much the same manner as previously reported.[2] A head rest of sponge rubber to eliminate direct vibration from the table is used instead of a metal clamp, and urethane combined with ether, is used for anesthesia in place of straight ether. This combination has definitely reduced operative mortality. Care is taken to maintain the animal's body temperature as nearly normal as possible.

There are three standard experimental conditions which must be considered before any modification of the conducting mechanism can be produced. These are an intact bulla, an opened bulla and one in which the septum has been removed. The complexity of the whole problem of bone conduction immediately becomes apparent when a change in response is noted between these three conditions. In a considerable series of experiments bone conduction tests have been made with a closed bulla; after the bulla is opened; and again after the septum between the bulla and middle ear has been removed. The following effects were noted. With an open bulla, and cotton in the opening, an increased intensity of response occurs over that obtained with an intact bulla; with septum removed there is a marked falling off in response. Of itself the septum would hardly be considered a sufficiently important route for normal bone conduction to cause such an effect by its removal. This is a constant finding, however. Fig. 3 illustrates the impairment in bone conduction which occurs



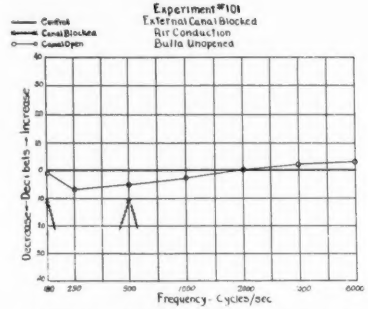
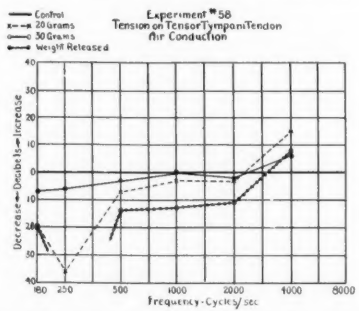


Fig. 5. Marked improvement of B. C. occurs with external canal blocked while A. C. is almost completely lost in an ear with intact bulla. Note return to control level. 8000 not transmitted in control test by B. C



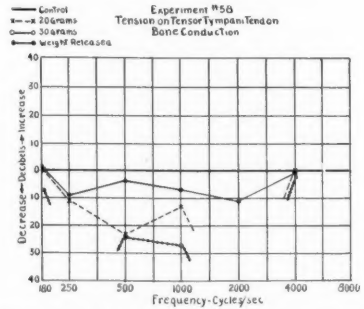


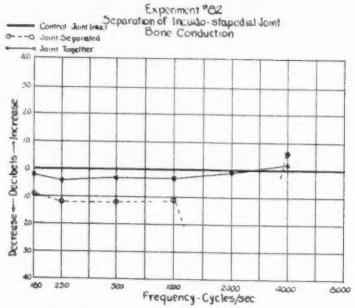
Fig. 6. Impairment of transmission is in direct proportion to amount of tension exerted on tensor tympani tendon. B. C. is affected more than A. C. Note lack of complete return of transmission for B. C. in comparison to return for A. C. when tension is released. Also fact that certain frequencies are actually lost by B. C. 8000 not transmitted by either A. C. or B. C. in control test.

in an open bulla after the septum has been removed, while air conduction remains unaffected.

Blocking external canal. The well recognized effect upon bone conduction of blocking the external canal has been studied in a small series of experiments. The method used was to pack the canal rather tightly with vaselin gauze, care being taken not to touch the drum. At first difficulty was experienced in obtaining the expected improvement. This was apparently due to the fact that the bulla had been opened before the procedure was carried out, for with an intact bulla bone conduction showed definite improvement. The reponse returned to normal when the gauze was removed and this effect could be repeated. Fig. 4 demonstrates the slight impairment which occurs with an open bulla, while Fig. 5 shows the improvement in bone conduction after the canal had been blocked in an ear with an intact bulla. However, air conduction in both instances was practically lost following blockage of the canal and returned immediately to its control level after the obstruction was removed. Kobrak found only a slight effect upon air conduction by blocking the canal with cotton, and variation depending upon the way in which the cotton was inserted. The vaselin gauze is obviously a more efficient block to air borne stimuli than dry cotton.

Tension on Tensor Tympani Tendon. Tension applied to the tensor tympani or stapedius tendons has a very definite effect upon air conduction. As previously reported,[2] a study of the function of these intrinsic muscles indicates that it is largely, if not entirely, protective. Kobrak has used their reflex contraction to study bone conduction in rabbits and felt that this reflex was an accurate measure of bone conduction threshold. Following tension, artificially exerted on the muscles, a very striking effect has been observed in the electrical response of the nerve to any bone conduction stimulus, analogous in every way to that observed for air conduction. With increasing tension bone conduction is more and more impaired until only one or two frequencies are transmitted, and following release the response returns practically to its normal level. Fig. 6, a typical example of this effect, illustrates the similarity of the response of both air conduction and bone conduction to extreme muscle tension. Under this condition both responses are definitely lowered, and with release of tension the response promptly returns to the control level.

Separation of incudo-stapedial joint. Separation of the incudostapedial joint will practically eliminate air conduction, which re-



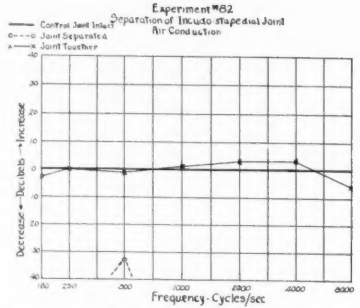
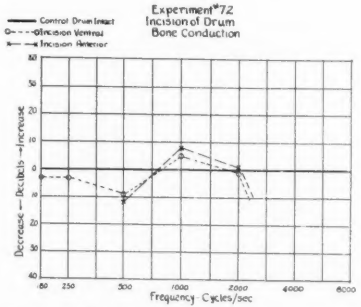


Fig. 7. Note almost complete loss of A. C. when the joint is separated. B. C. is only slightly impaired. Both A. C. and B. C. return to control level when the joint surfaces are placed in contact again. 8000 not transmitted by B. C. in control test.



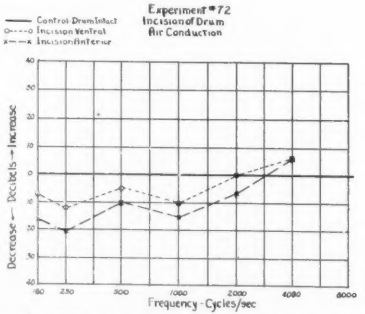


Fig. 8. Low tones by both B. C. and A. C. show impairment after incision of the drum, the amount of impairment depending on the size of the incision. The two low frequencies were entirely lost by B. C. when the incision of the drum was enlarged.

turns to the control level when the articulating surfaces are allowed to come into contact with each other. This type of experiment has been described as a "reversible lesion." This series of experiments, previously carried out on air conduction, has been repeated and the bone conduction response studied. Fig. 7 illustrates the effect obtained. Air conduction is almost competely lost, while bone conduction is definitely lowered. Return of the articular surfaces in apposition to each other produces the usual return of response for both air and bone conduction. In these experiments it may be difficult to divide the joint without damaging either the process of the incus or stapes. If such damage is done it is impossible to restore apposition, and the lack of continuity may be corrected by inserting a bone chip between the two ossicles. When this is done the return to control level is secured equally well.

Incision of drum. In a previous report the effect upon air conduction of different sized incisions of the drum was detailed. It was felt that the lowering of the electrical response was very much less than might have been expected. This experiment has been repeated and the effect of incisions of the drum upon bone conduction studied. In Fig. 8 it can be seen that following incision a striking effect is produced upon bone conduction at the lower and higher frequencies. With large incisions the high and low frequencies are lost entirely. The frequencies 1,000 and 2,000 are practically unaffected. In this experiment the response by air conduction shows a much more marked impairment than that previously reported, but all frequencies are still transmitted by air conduction even with large incisions.

Albino animals. Howe and Guild[23] reported the absence of electrical response in congenitally deaf albino cats. These observations have been confirmed and in addition there was found a complete absence of response both by air conduction and bone conduction in congenitally deaf Dalmation puppies.

DISCUSSION

The experimental studies on bone conduction reported up to the present time have concerned themselves chiefly with the establishment of the primary pathway for bone conduction. Efforts have been made to determine whether the principal rôle must be allotted to the petrous portion of the temporal bone or to the ossicular chain. Theory and the interpretation of experimental data seem fairly equally divided in the matter, so that nothing more definite can be

assumed at the moment than that each is important. Wittmaack[5] asserts very definitely that the ossicular chain is the primary pathway while other authors with equal certainty regard the direct route through the bone as of greater importance. Krainz,[6] in a beautifully designed series of experiments on the cadaver, accepts the two routes and analyzes the effect of middle and outer ear changes upon the phase relation between the two series of vibrations set up by the stimulation. Kobrak's method of study represents a very distinct departure from earlier types of experimentation. His investigation of the tensor reflex leads him to believe that certain conditions actually increase bone conduction, qualifying his opinion somewhat by advancing the statement as an interpretation of observed experimental findings. The masking effect of noise in normal as compared to deaf individuals is a matter of considerable interest in the conductive type of deafness, but here again opinion is at variance. Knudsen and Jones[7] found no difference in examining cases of conductive deafness and those of normal hearing in a sound proof room, while Kobrak believes that the increase in bone conduction is real.

The experimental data outlined above is offered more as a series of observed facts than as an effort to establish any theory of bone conduction. While admitting that the technical difficulties of this electrical method of study increases the possibility of error, nevertheless it is felt that the data offered is of considerable significance. Measurements by a large group of investigators have established the importance and accuracy of action potentials in the acoustic nerve.

In earlier experiments at the Johns Hopkins Laboratory, Howe repeatedly demonstrated the fact, which Krainz described, of an actual increase in bone conduction after the middle ear is filled with mercury. Krainz's explanation of the phenomenon is that there occurs an increase in the effective mass of the ossicular chain. If this is valid the same reason undoubtedly holds true in the presence of a gross infection involving the middle ear and bulla. In the occasional instance where such a condition is found in the experimental animal bone conduction is definitely increased.

With the exception of the blocked external canal the invariable effect of all experimental procedures carried out by us was a decrease in the response to a constant intensity of bone conduction stimulation. In this connection also it must be realized that bone conduction and air conduction are always affected in the same direction if not to the same degree. All investigators have emphasized the fact that

bone conduction changes can be compared qualitatively but never quantitatively to air conduction. The point of interest, therefore, lies in the fact that changes in response manifest themselves simply as improvement or impairment and not the extent to which these changes develop. A rather remarkable fact is that with changes in the conductive system certain frequencies may be completley lost by bone conduction.

Incision of the drum with the subsequent reduction in bone conduction response far exceeding the reduction in air conduction is difficult to reconcile with the opposite effect produced in the reversible experiment of dividing the incudo-stapedial joint. Here the stapes has been completely isolated from the conductive system, and air conduction is completely lost at low intensity of stimulus, yet bone conduction is relatively unaffected. The return to normal of both responses when contact of the joint surfaces is restored is a constant finding. The factors concerned in the difference between these two effects can only be hypothecated at the moment. The element of change previously referred to must be considered in any discussion of these observed phenomena, namely, the fact that in all experiments upon middle ear structures the bulla must be open and the septum at least partially removed. .

In the tension experiments the change in response for both air and bone conduction is relatively of the same degree and in the same direction. One point of differentiation develops, however, that in bone conduction the frequency range from 500 to 4,000 d.v. is affected less than air conduction. Fixation by tension would seem most likely to exert its effect upon the conductive mechanism and not in any way upon intralabyrinthine pressure. The tension in every instance fixes the stapes by drawing it slightly away from the oval window, and even were the fixation in the opposite direction no pressure change would be produced, due to the elasticity of the round window. Indeed, air conduction itself is a control in this particular question for, as has been demonstrated by measured changes of intralabyrinthine pressure, air conduction is not affected by marked increase in this fluid pressure.[8]

It is obviously impossible from the data to draw any conclusions as to the rôle played by phase change, impedence, mass effect or elasticity of the conductive mechanism in the effects produced upon bone conduction by the various experimental procedures. On the other hand, the results obtained indicate very definitely that the con-

ductive mechanism of the ear in experimental animals plays a major part in transmitting bone conducted stimuli to the sensory cells of the cochlea. So important is this particular route that transmission by bone conduction at a certain standard intensity of stimulus can be completely eliminated by experimental procedures carried out upon the middle ear structures. The demonstration of this fact depends entirely upon elimination of the possible sources of error emphasized above. The fact that response by bone conduction can be so changed attests to the effectiveness of the precautions taken.

CONCLUSIONS

1. Bone conduction in the cat, determined by measurements of change in electrical potential in the auditory nerve, is increased by blocking the external canal. This is only true if the bulla is intact.

2. Both air conduction and bone conduction are affected by incision of the tympanic membrane, the latter to a much greater extent than the former.

3. Divison of the incudo-stapedial joint causes a loss of air conduction and an impairment of bone conduction.

4. Fixation of the ossicular chain by tension applied to the tensor tympani tendon impairs both air and bone conduction.

5. The conductive mechanism of the ear has a major rôle in the phenomenon of bone conduction.

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LXV

END-ORGAN DEAFNESS IN DOGS DUE TO THE APPLICATION OF CERTAIN CHEMICALS TO THE ROUND WINDOW MEMBRANE*

E. P. Fowler, Jr., M.D.

AND

T. W. Forbes

New York

In a previous communication we have demonstrated that there is a progressive loss of action potentials in the cochleæ of cats whose round windows have been treated with electrolytes such as sodium chloride, calcium chloride and quinine dihydrochloride.[1,2] In this paper we intend to show similar findings in dogs, and to point out certain clinical implications.

The technic used was briefly as follows: young dogs of the same litter were conditioned to sound by a modification of the method of Culler.[3] The dogs were placed in a box with a grill under the right forepaw, the receiver of a 2A audiometer was held first to one ear and then to the other. The operator, out of sight of the dog, worked the audiometer and observed his reactions. A mild shock was administered to the right forepaw a few seconds after a tone was sounded in the receiver. The dogs very quickly learned that they could avoid the shock by lifting the right forepaw. The intensity of the sound was then cut down until the dogs no longer lifted the paw, and a series of threshold readings were taken over a period of several days for each ear (Fig. 1). When the conditioning was complete and a reliable threshold audiogram was obtained, the animals were placed under nembutal anesthesia and the bullæ of the middle ears exposed through a midline incision in the neck. Using the Wever and Bray technic one electrode was placed on the promontory and another in the neck muscles of the dog and wires run to another room where were housed the amplifier and an observer with head phones. The minimum amplifications needed to hear a harmonica and a Galton whistle were recorded by the observer for each ear. The ear with the best record was then selected for the experimental ear, and a crystal

*From the Departments of Otolaryngology and Pathology, Columbia University, and the New York State Psychiatric Institute, under grants from the American Otological Society and the Hayden-Coakley Fund for Research in Otolaryngology. Mrs. T. W. Forbes did all of the routine work in connection with the conditioning of the animals.

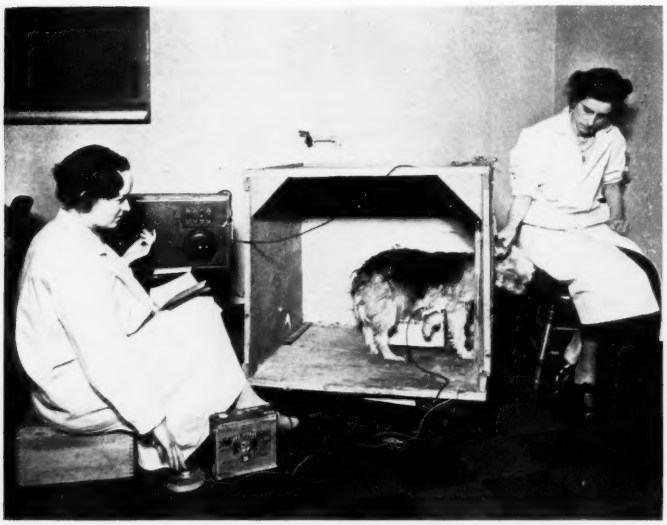

Fig. 1. Method of conditioning dogs.

of sodium chloride or calcium chloride was placed in the niche of the round window. The chemicals were allowed to remain until a marked loss of cochlear potential was recorded. The other ear was used as a control. When the desired loss of cochlear potentials had been produced the wound in the neck was closed. Two days after the operation the dogs were well enough to be put back in the conditioning box. In cases in which sodium chloride had been used an actual hearing loss was apparent in the experimental ear. After a series of retests by the conditioned reflex method the dogs were again operated upon and a check by the Wever and Bray method was carried out. Surprisingly enough, rarely was an infection present to nullify the results. The dogs were then perfused through the aorta with saline and formalin for intravital fixation and the temporal bones removed for serial sectioning.

RESULTS

Approximately the same results were obtained for dogs as for cats when crystals of sodium chloride or calcium chloride were placed on the round window: i. e., there was a progressive loss of cochlear potential as obtained from the promontory. Histologic specimens of the dogs treated with sodium chloride show similar lesions to those found in the cats—that is, when the dogs were killed two to eight

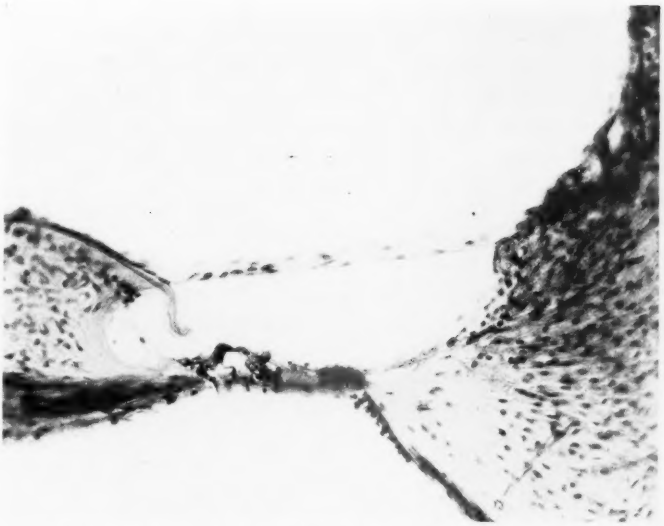

Fig. 2. Nearly total destruction of organ of Corti in dog on whose round window membrane crystals of sodium chloride had been placed.

Fig. 3. Near top of first turn of cochlea of a dog whose round window had been treated with crystals of sodium chloride. There is swelling of sustentacular cells, atrophy of external sulcus cells and absence of the second and third outer hair cells of the organ of Corti.

days after sodium chloride had been placed on the round window, there was marked destruction of all the cochlear elements and sometimes of the nerve elements in the basal coil of the cochlea. In the upper coils there was progressively less and less destruction until nothing abnormal was apparent as the apex was approached. The end organs within three-quarters to one-half a turn from the apex usually appeared entirely normal. The lesions in the second turn were usually similar to those found by Lurie[4] in his stimulation deafness experiments; that is, in our dogs there was complete or partial destruction of the outer hair cells or occasionally destruction of all of the hair cells, while in the upper part of the lowest turn there was often destruction of the sustentacular cells as well as of the hair cells. In addition, whenever there was the slightest change in the outer hair cells there were always changes in the external sulcus cells. The observations of destruction in the external sulcus cells in our experimental high tone deafnesses fits in with the observations of Crowe, Guild and Polvogt,[5] who found similar changes in humans with high tone deafness.

With calcium chloride there was a marked drop in cochlear potential as measured by the Wever and Bray apparatus. This was similar and roughly equal to the drop found with sodium chloride and the drop found with calcium in cats. However, when the dogs were reconditioned it was found that they had no loss in hearing, in spite of the fact that in the retest by the Wever and Bray method they were found to have maintained a loss of cochlear potential. In the histologic sections there was complete destruction of the organ of Corti at the very lowest part of the lower turn, there were slight changes in the hair cells up to the end of the first turn, and there were changes in the sustentacular cells almost as far up as the changes in the hair cells. But there was destruction of the external sulcus cells almost to the level of the end of the second turn of the cochlea. We have too few animals to be conclusive at present, so that we hesitate to commit ourselves on an interpretation of this finding as yet.

COMMENT AND CLINICAL SIGNIFICANCE

From the findings of Osterhout,[6] who worked with unicellular organisms and with plant cells, it would seem the slightly hypertonic concentrations of calcium should have an exactly opposite effect from similar concentrations of sodium in changing the permeability of cell membranes and so affecting the release of cell potentials. If a more refined apparatus is used, perhaps the key to the different effect of calcium on hearing will be ascertained.

Our experiments indicate that a place theory for hearing is correct, inasmuch as the high tones were most affected by chemicals seeping through the round window and the loss was progressive from the high tones to the low tones, depending upon the length of time the chemicals were in contact with the round window. It would seem that those who have postulated that the high notes are localized in the lower turns, while the low notes are localized in the upper turns of the cochlea, are correct. Our experiments further indicate that the hair cells and possibly the external sulcus cells are essential for the picking up of electrical potentials by the Wever and Bray method.

This series of experiments was included in the Symposium on Bone Conduction, because, after all, a loss in cochlear potentials as recorded by the Wever and Bray method, is really comparable to a loss in bone conduction; therefore, what we have produced with our chemicals is a so-called "nerve deafness," more specifically a primary end organ deafness.

Certain types of nerve deafness have never been satisfactorily explained; for example, the nerve deafness which occurs in some patients with chronic suppurating ears. It seems possible that the calcium phosphate from degenerating bone of the mastoid and the toxic materials produced by bacteria in the exudate in the middle ear might poison the lower turns of the cochlea through the round window in these cases, just as our experimental chemicals have done.

Our experiments suggest a possible therapy for high-pitched tinnitus. It might be possible to instill the correct concentration of sodium chloride through the drum so that the solution would lie in the inferior part of the middle ear and seep through the round window to injure the cells of the end organ of the lower turns of the cochlea. Irritation of these cells may cause a high-pitched tinnitus; and if so and if we could destroy these cells without damaging others, the tinnitus might stop. One cannot help wondering if the effects reported by Albert Gray[7] with thyroxin may not be due to some effect such as the one postulated above. He reports that the chief improvement of hearing in his cases was due in large part to the removal of the masking effect of a tinnitus.

FURTHER WORK

We hope to try thyroxin on the cochlea of the cat, and see if it produces injuries of the lower turns analogous to those found with other electrolytes. If it does, simpler and cheaper chemicals,

such as sodium chloride, should be used for this type of work. Our equivocal findings with calcium need more experimental data and more delicate apparatus than we have had available.

A more obvious need, however, is the study of the effect of the common chemicals used in the ear therapeutically. Our early experiments seemed to indicate a slight effect with boric acid. We hope to study this further and in addition to try such common agents as Sulzberger powder, silver nitrate, bicarbonate of soda, etc., and see if they too progressively poison the finer mechanism of the cochlea as they seep through the round window.

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LXVI

CONGENITAL PAPILLOMA OF THE TRACHEA—DIVERTICULUM OF TRACHEA—RUPTURE OF DIVERTICULUM OF TRACHEA INTO INNOMINATE ARTERY*

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The patient I am reporting on was admitted to Babies' Hospital, Columbia Medical Center, March 4, 1935, at the age of two months. The chief complaint was choking when taking the bottle and trouble in breathing. The baby was born at full term, normal delivery; no asphyxia or cyanosis. There were supernumerary digits on both hands attached to the fifth finger. The breathing had been noisy from birth with crow and retracture. The child got along fairly well, although the respiration continued noisy up to three days before admission.

The stridor in the last three days has been much more severe; there has been more choking on feeding. She was quiet in sleep, although it was noticed that her nostrils dilated.

Physical examination showed a well developed and well nourished child, in great respiratory difficulty. Labored inspiration, marked retraction, stridor. Cyanosis becomes worse when crying. Lungs, clear. No obvious abnormalities of the pharynx.

The laboratory findings were: Hemoglobin, 103 per cent; white blood count, 15,000. Throat culture was negative for diphtheria; streptococcus hemolyticus predominates. Urine was normal. The tuberculin test was negative. Culture from the tracheotomy tube showed staphylococcus aureus hemolyticus predominating, with a few streptococcus hemolyticus. Kahn test was negative.

*Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

On account of the extreme dyspnea, the patient was put into a tent supplied with a mixture of helium and oxygen approximately 80 and 20. This gave the child marked relief, although there was still slight respiratory distress. After trial of a week of helium, since the child became dyspneic whenever it was taken out for changing and other care, it was felt that some surgical procedure might be necessary. Up to this time it had been thought that the difficulty in breathing was caused by some lesion in the larynx. On bronchoscopy, however, the larynx was found to be normal. The obstruction was lower down in the trachea, about one-half inch above the bifurcation. With the bronchoscope still in place a tracheotomy was done, and an ordinary tracheotomy tube No. 1 was inserted. It was then found that this gave the child no relief and on further investigation it was found that only by pushing the bronchoscope actually down to the bifurcation was relief secured. The bronchoscopic appearance was of a soft mass pushing into the lumen from in front and to the left. The child was returned at once to the helium and oxygen tent. A special long cane shaped tube was secured and inserted. This tube gave complete relief from the dyspnea. After this the patient developed a pneumothorax on the left side, which was absorbed.

The subsequent course was, on the whole, downhill most of the time, although there were intervals when she seemed to improve fairly steadily. Difficulty was had in keeping the tube sufficiently deep to reach beyond the obstruction. Also, it was found that the end had a tendency to slip into one or the other of the main bronchi, aerating that particular lung but causing a massive collapse in the other. A great deal of mucus was secreted, requiring frequent suction. This was often blood tinged. Because of this show of blood the development of granulation tissue was feared, and the use of tubes of different lengths was attempted. This was not successful because a short tube did not relieve the obstruction, and a longer tube slipped into one of the main bronchi, leaving the other lung without air. On May 7th, because the respiratory difficulties were becoming worse, it was felt that radiotherapy should be attempted, in view of the extreme probability of obstructive granulation tissue about the tube near the bifurcation of the trachea. On May 10th, when the tube was being withdrawn in order to change it, a gush of fresh blood came from the tracheotomy wound. The tube was immediately replaced and suction instituted. The hemorrhage gradually subsided, and in about one-half hour the mucus was merely pinkish. The next night, while the patient was lying quietly in bed, there was sudden profuse hemorrhage from the tracheotomy wound. Death was immediate.

For our purposes, only the autopsy findings of the examination of the larynx and trachea will be given. The larynx was normal. The tracheotomy wound was .5 by .4 centimeters in diameter. It had clean granulating edges. Extending for a distance of two and four-tenths centimeters above the bifurcation on the anterior walls of the trachea was an elliptical, raised, definitely outlined area one centimeter wide, which had an irregular surface. Its appearance suggested a papilloma. It extended into the right main bronchus as far as its bifurcation. A small round hole two by three millimeters in diameter, with red irregular torn edges, appeared two millimeters below the upper border of the raised area. This opening led into the innominate artery just where it came off the aortic arch. It was one millimeter wide, had ragged edges, and easily admitted a probe. The raised area extended to the posterior trachael wall over a distance of one and two-tenths centimeters. On section at the bifurcation the posterior tracheal wall is seven-tenths of a millimeter thick. The lymph nodes were normal. The microscopic examination of tissue showed:

Trachea 1. Sections through the anterior wall of the thickened lower end of the trachea shows metaplasia of the lining epithelium, so that there are many layers of squamous cells between the papilliform elevations of the mucosa, and fewer layers over the broad tops of the papillae. Polymorphonuclear infiltration is mild within these elevations, but becomes dense in an area of mucosa denuded of epithelium, in which, however, the irregularities of the surface still persist. This part of the tracheal wall is thickened with granulation tissue in the mucosa and submucosa. The adjacent cartilage is normal, but cellular infiltration extends between the mucous glands and even to the outer layers of the perichondrium in places. Many eosinophilic cells are present. Three small lymph nodes in the outer coat show germinal centers in which there is edema and some necrosis. The sinuses are wide and not filled with cells.

Trachea 2. This section is through the posterior wall at the level of the roughened mucosa. Here the finger-like processes on the free surface are much higher and more branched, and the epithelium over their lower and adjacent surfaces still ciliated columnar in type, though squamous over the apex. Polymorphonuclear infiltration is less marked in this part of the trachea, but in part of the section it is intense and involves all the coats, even the muscle.

Trachea 3. Serial sections were made through the opening between the trachea and the innominate artery. Here again the meta-

plasia, the finger-like elevations on the mucosa, the intense inflammatory infiltration of the tracheal wall, even extending into the cartilage through the perichondrium, are apparent. In the inflamed lip of the opening of the trachea the epithelium dips down beyond the mucosa, and this adds to the evidence of the presence of a small diverticulum. Then the areolar tissue between the trachea and the large vessels is here shown to be most densely packed with polymorphonuclear leucocytes, and this infiltration goes on into all the coats of the vessel. The floor of the rupture in the artery shows polymorphonuclear infiltration and necrosis and irregular tearing of the elastic fibers of the media. The intima beyond the rupture shows thickening, polynuclear infiltration and fibrin on the free surface.

SUMMARY

This most interesting case presents three distinct problems: First, the raised, roughened and thickened area above the tracheal bifurcation; second, the rounded opening in the upper part of this area, with its thickened edges; third, the erosion into the innominate artery. As to the first: The author is certain that he saw a roughened place on the tracheal lining when he did the bronchoscopy on March 12th. That observation, and the fact that the child had had attacks of cyanosis and noisy breathing in the newborn period, would seem to show that the mass just above the bifurcation was a primary lesion present at or soon after birth, and not of secondary inflammatory origin. That inflammation increased its size is undoubted, but the papillomatous character of the lesion was apparently the primary thing. As to the second point: The round shape of the opening would be difficult to explain on the ground of erosion by the circular end of the tube, which could, of course, only press with one edge, and so make a linear or curved tear. The hole, with its rounded, thickened edges in the papillomatous area, is adequately explained by the assumption that it is the opening into the congenital diverticulum of the trachea, as suggested by Dr. von Glahn. The diverticulum in this case must have been very short, possibly only two or three mm., but the depth at which epithelial cells are seen in the edge of the tracheal opening, below the inflamed area, makes this assumption plausible. The inflammatory infiltration may obscure the lower, blind end of the diverticulum, which is not demonstrable. This is unfortunate. An article by Chiari in Ziegler's Beitrage, 1889, 5:329, explains and illustrates tracheal diverticula. As to the third point: Serial sections show the extension of the inflammation from the tracheal wall into the areolar tissue between it and the vessels, into the

adventitia and other coats of the innominate artery. The inflamed diverticulum and the inflamed arterial coat, encountering the constant pressure of the hard metal tube with each arterial beat, over a period of two months, led to the rupture and fatal hemorrhage probably.

103 East 78th St.

LXVII

COSTOPHRENIC BRONCHOSCOPY FOR PINS DEEP IN THE TRACHEOBRONCHIAL TREE*

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When a long foreign body of small diameter and pointed at one end, like a pin, enters the respiratory tract it has a tendency to penetrate to the lowest possible location in the trachebronchial tree, lying point up. If it happens to lodge in the main stem bronchus previous to bronchoscopy, the introduction of the bronchoscope widening the bronchus in combination with the ratchet like movement of the pin due to the point in contact with the bronchial wall and the respiratory movements of the bronchus contracting and elongating, the tendency will be for the pin to descend lower and lower until it lodges in a very small branch bronchus at the periphery of the lung.

The problem of removal then becomes a complicated one, because the pin has passed beyond the point where it can be seen through a tube.

It is impossible to have a bronchoscope, carrying a light, small enough to penetrate to the branch bronchi at the periphery of the lung so it becomes necessary to use a small tube without a light and depend upon one's sense of touch and the help of the fluoroscopist in passing it. The tube must be passed with the utmost gentleness and caution to avoid traumatism and to find the branch in which the foreign body is lodged.

The fluoroscopist must needs be an expert in rendering assistance with the double plane fluoroscope. The latter is an absolute necessity, otherwise the forceps may be ready to adjust to the point of the pin in one plane but not near the point in the other plane. The fluoroscopist must guide the closed forceps first in one plane and then in the other plane until the adjustment is perfect and the forceps can be opened to grasp the point and bring it into the tube mouth.

*Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

REPORT OF CASES

CASE 1.—A little girl, two and one-half years of age, removed a straight pin from her doll's dress and put it into her mouth. She began to choke and cough, informing her parents that she had swallowed a "needle." She was brought to the Presbyterian Hospital December 19, 1935, apparently suffering from no symptoms, and the roentgenogram showed the pin in the right lower lobe bronchus.

Two fluoroscopic bronchoscopies were attempted, which were unsuccessful because the double plane fluoroscope failed to function, consequently it was necessary to have the fluoroscope rebuilt and this necessitated considerable delay. For two months the child had no symptoms, playing about the ward with the other children. The roentgenogram, taken February 20, 1936, showed the pin lying at the base of the right lung close to the diaphragm and along the inner margin of the lung.

Without anesthesia, under the guidance of Dr. George W. Grier, with the double plane fluoroscope, I passed the Chevalier Jackson costophrenic bronchoscope to the point of the pin and removed it in 25 minutes, 39 seconds.

There was no reaction following bronchoscopy, but the following day the child developed an eruption on the chest which was rather puzzling. It proved to be German measles and delayed her discharge during the period of quarantine.

CASE 2.—A little girl of four years, during the absence of her parents, climbed up to the dresser to get a pin to put in her doll's dress. Her father was punishing her for this and noticing the pin in her mouth, told her to remove, but with a sob she aspirated it. She was admitted to the Presbyterian Hospital April 1, 1936, suffering no symptoms. The first roentgenogram showed the pin in the left main stem bronchus below the bifurcation, point up and round head down. The following day, upon introducing the 5 mm. bronchoscope into the left bronchus, the pin receded rapidly and a roentgenogram showed it at a point much lower down in the bronchus, point up.

April 5th, without anesthesia, under guidance of Dr. George W. Grier, with the double plane fluoroscope, I passed the 5 mm. bronchoscope, located the pin, but had difficulty in applying the forceps to grasp the point. Withdrawing the 5 mm. tube, I passed the Jackson costophrenic bronchoscope, with Dr. Grier's assistance, and removed the pin in nine minutes. There was no reaction and the child was discharged in forty-eight hours.

In the first case, with the pin at the periphery of the lung, the use of the costophrenic bronchoscope was the only method by which the pin could be removed, and in the second case the small diameter of the costophrenic bronchoscope facilitated the application of the forceps to the point of the pin. In order to use this type of bronchoscope which carries no light, one must have the assistance of a fluoroscopist expert in the use of a properly functioning double plane fluoroscope, which should be equipped with a time clock connected with each plane to record the time of exposure.

121 UNIVERSITY PLACE.

LXVIII

PAPILLOMA OF THE TRACHEA: REPORT OF A CASE*

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The patient was a boy, seven years of age. He complained of hoarseness and difficulty with breathing.

The family history was negative. His personal history showed that he had measles and whooping cough and no operations, and that generally his health had been fairly good.

Two months previous to admission to the Jefferson Hospital he gradually developed hoarseness and dyspnea, which progressed and became quite marked, regardless of medical treatment at his home. He was admitted to the bronchoscopic clinic August 26, 1935, for diagnosis and treatment.

Physical examination: The patient was a well nourished male child. There was very little, if any hoarseness. There was marked difficulty with breathing, both on inspiration and expiration, and a suggestion of cyanosis. The eyes and ears and mouth were negative. The nasal mucosa was congested and there was increased secretion in both nostrils. The teeth were in fair condition and the tongue clean. The tonsils were markedly hypertrophied. The cervical lymph nodes were palpable on both sides. The trachea was in the midline. Chest examination revealed rough breath sounds, evidently transmitted from the trachea.

X-ray examination, May 28, 1935, revealed no evidence of opaque foreign body in the region of the neck. The trachea occupied a normal position in both the anteroposterior and lateral planes. There was no evidence of opaque foreign body in the lungs. The pulmonary markings were slightly increased in the right lower lobe, otherwise the lungs had an essentially normal appearance. The heart and diaphragm shadows were normal.

Tracheoscopy, May 28, 1935, revealed that the larynx was normal above the level of the vocal cords. Quite a large mass of papillo-

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*Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

matous material almost completely obstructed the subglottic area and extended well down into the trachea. The masses were readily detached by reaming the subglottic larynx and the trachea with the bronchoscope and removing masses by means of an aspirating tube.

The laboratory reported the specimens to be papillomata. The secretion below the papillomata contained micrococcus catarrhalis, staphylococcus albus, pneumococcus and streptococcus hemolyticus.

Progress of the case: Most of the dyspnea disappeared after the tracheoscopy. There was a somewhat croupy cough, which promptly cleared up.

At the second tracheoscopy, four days later, a small mass beneath the left vocal cord was removed by means of an aspirator. No papillomata were seen in the trachea.

The patient was discharged one month after admission. The tracheoscopy, just prior to discharge, showed that the larynx and trachea were free of papillomata.

For three months following his discharge from the hospital, the patient's condition continued good with no difficulty in breathing or huskiness of voice. He then developed chickenpox, which was accompanied by dyspnea, most marked when lying down, and a slightly husky voice.

He was readmitted to the bronchoscopic clinic January 6, 1936, and several masses of papilloma were found on the anterior tracheal wall, about one centimeter below the vocal cords. These were removed, using the tip of the bronchoscope. The larynx and lower trachea were free from growths.

A very small mass of papilloma was removed from the same site one week later.

The patient was discharged five weeks later, February 14, 1936, with nothing to suggest recurrence.

COMMENT

Papilloma of the larynx is a common neoplasm in children, whereas papilloma of the trachea is rare.

In a series of 314 cases of papilloma treated at the Jefferson Hospital Bronchoscopic Clinic since 1917, 312 were papilloma of the larynx and two (excluding this case) were papilloma of the trachea.

The history of hoarseness prior to admission was somewhat misleading and probably was due to inflammation of the larynx, as none

of the masses were near enough to the edges of the vocal cords to prevent good approximation during phonation.

The papillomatous masses were removed very easily by simply shearing them off with the tip of the bronchoscope and picking them up with the end of an aspirating tube.

The growths probably will recur, and direct removal is the only method to be employed in their treatment.

1923 Spruce St.

LXIX

HEART-BLOCK PERIODICALLY INDUCED BY THE SWALLOWING OF FOOD IN A PATIENT WITH CARDIOSPASM (VAGOVAGAL SYNCOPE)*

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AND
BERNARD A. SCHWARTZ, M.D.

CINCINNATI

In 1934, Soma Weiss and Eugene Ferris described a peculiar form of syncope, which they determined was of reflex origin, with the entire reflex arc located within the vagus system. They, therefore, termed the symptom complex "Adams-Stokes Syndrome With Transient Complete Heart Block of Vagovagal Reflex Origin,"[1] or briefly, "Vagovagal Syncope."[2] In vagovagal syncope the afferent impulses originate at some point in the sensory ends of the vagus and are transmitted to the vagus center, from which they are shunted through the efferent motor fibers, terminating in the heart, causing slowing of the heart with complete block.

The patient of Weiss and Ferris, in whom this phenomenon was observed, gave a history of syncopal attacks covering a period of ten years. The attacks were usually incited by the taking of food. Between attacks the patient felt perfectly well. Regarding his condition as incurable, the patient had attempted suicide.

Physical examination revealed no gross abnormalities aside from a small diverticulum of the esophagus. The authors assumed that the sensory terminations of the vagus within the diverticulum were excited by ingested food, and that this stimulus was transmitted reflexly along a vagal arc to the heart. Accordingly, an experiment was made by introducing an inflatable balloon into the esophagus at the level of the diverticulum. When the balloon was inflated an immediate and complete heart block ensued. The block disappeared when the balloon was deflated. In this manner an Adams-Stokes syndrome

*From the Department of Otolaryngology, College of Medicine, University of Cincinnati.

Read before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

could be produced at will. When atropin sulphate was administered, inflation produced no effect upon the heart. Similarly, when either the right or left vagus was blocked by novocain injection, inflation no longer inhibited the heart, proving the reflex nature of the phenomenon.

The authors collected two additional cases of vagovagal syncope and found two authentic cases reported in the literature. Of the latter, that of Gluch[3] presented a thoracic tumor combined with a paralyzed left vocal cord. At autopsy a sarcoma was found enmeshing the left vagus nerve, and it was thought that the irritation of the vagus by the tumor had excited the reflex. Weiss[2] has recently observed two similar tumor cases, also a case in which an Adams-Stokes syndrome was excited each time a bronchoscope was introduced into the left bronchus of a patient.

The diagnosis of transient neurogenic heart block is only justifiable in cases in which structural lesions within the heart can be excluded.

REPORT OF A CASE

CASE 1.—In November, 1935, Mrs. H., 55 years, came under our care. She complained of occasional fainting spells while partaking of food.

At the age of four years she had had diphtheria. From her ninth year on she had had fainting spells at long intervals, but these ceased about fourteen years ago. (These syncopal attacks, followed by vomiting, were unlike her present ones, and were not epileptiform in nature, although they were preceded by an aura manifested by tingling in the feet.) She had had "nervous exhaustion," associated with fainting spells, in 1924. In 1931 she had undergone a hysterectomy and ovariectomy.

In May, 1929, the patient began having trouble with deglutition, and would have a sense of weight and fullness under the sternum while partaking of food. Vomiting never occurred. Roentgenograms of the alimentary tract had been taken in 1931 and 1935. These revealed what was described as a spastic condition of the esophagus.

In September, 1934, she began having peculiar fainting spells. These attacks occurred at irregular intervals while at the table partaking of food of any kind, including water. The spells came on, sometimes at the beginning of the meal, sometimes toward the end. The attacks were sometimes preceded by a full sensation at the pit of the stomach, ascending toward the throat. (If belching occurred, there was usually no fainting.) Her associates stated that her face would flush and then grow pale at the time of the attack and she herself had made the same observation in a mirror. During the spells things would grow dim before her eyes but there was no vertigo. The duration of the attack was momentary. On two or three occasions she completely lost consciousness. On repeated occasions, immediately after the fainting spells, she noticed that her pulse had stopped and then began to beat slowly and intermittently. At the time the patient presented herself she had been taking ephedrine, gr. 3/8 (.024), before each meal for a period of four months, and had been almost free from syncopal attacks. During the past

Fig. 1. Typical roentgenogram showing cardiospasm with a dilated esophagus.

year she had lost considerable weight (lowest weight, 112 pounds; best weight, 136 pounds).

Physical Examination.—Examination of head, chest and abdomen was essentially negative. The heart appeared normal in size and configuration.

Vascular System.—There were no thrills palpable over the precordium. The heart sounds were of fair tone, the rate 75-90, and the rhythm regular. No murmurs were heard. The blood pressure was 125 systolic; 80 diastolic. The radial vessels were not sclerotic. Pressure on the right carotid sinus caused a slowing of the pulse from 70 to 40; pressure on the left carotid sinus reduced the pulse from 76 to 60. Pressure on the eyeballs produced no effect on the heart rate.

FLUOROSCOPY AND ROENTGEN EXAMINATION

Esophagus.—X-ray examination of the esophagus, made fluoroscopically and with films, showed a definite delay in the passage of the barium mixture with

increased peristaltic activity. The esophagus was considerably dilated and narrowed to a smooth spindle at the diaphragm, presenting a typical picture of cardiospasm (Dr. William Doughty). (Dr. William Wasson) (Fig. 1.) Revision of the films taken in 1931 revealed evidence of the same condition. Heart—Fluoroscopic examination of the heart showed it to be of normal size and action. The aorta was not widened.

Electrocardiography.—November 11, 1935. Patient had omitted taking ephedrine for seventy-two hours. The electrocardiographic reading was practically normal. The patient was then given food, following which the electrocardiographic tracings, aside from a slight sinus arrythmia, remained essentially negative. This negative result was assumed to be due to a prolonged ephedrine effect. Four days later another electrocardiographic examination was made. The tracings (before food) showed a rate of 75 and a conduction time within normal limits. (Fig. 2.) Food was then taken by the patient. In about five minutes her face blanched and her eyes stared. The electrocardiogram revealed a two-one auricular ventricular block, followed by four normal cycles (Fig. 3). This phenomenon occurred five times in sequence and twice thereafter (Fig. 4). During each ventricular standstill the patient's face blanched, her eyes stared, and she complained of faintness. Then the dominant rhythm was re-established.

Diagnosis.—From the history, the x-ray findings, and the electrocardiograms in this case, it was decided that no organic heart condition was responsible for the phenomenon noted. The symptom complex was explained on the supposition that the ingested food either distended or excited peristaltic waves in the dilated esophagus (spastic at the cardia), and that peristalsis stimulated the sensory vagus endings in the esophageal wall, exciting a vagovagal reflex which inhibited the heart. Dr. Ferris, who saw this patient with us, agreed with this diagnosis.

Treatment.—It was felt that if the cardiospasm could be permanently relieved, the source of the reflex would be abolished and the vicious circle broken. It was therefore decided to employ bouginage with Hurst's mercury filled bougies, but it was feared that the introduction of the bougie might excite the vagal reflex. Therefore (December 28, 1935) a prophylactic dose of ephedrine sulphate, gr. 1/12 (.005), was administered hypodermatically, and about one minute later a 28 Eng.-46 Fr. bougie was passed into the stomach. There was slight resistance at the cardia. The patient grew slightly pale, but there were no other untoward symptoms. Some undigested food and fluid came up with the tube.

Bouginage was first practiced at bi-weekly intervals. Ephedrine medication was discontinued. The patient began eating meat for the first time in four months. During the first month's treatment the patient had two brief syncopal attacks while eating. By the end of the month the patient was able to pass the bougie herself. During the following month the patient introduced the bougie every other morning before breakfast. She was free from subjective heart symptoms, and food seemed to enter the stomach promptly. She began to gain in weight. During the following two months the tube was passed on alternate days. Occasionally a larger tube (32 Eng.-52 Fr.) was used.

At the present time the patient occasionally notices signs of heart block, but of less severity and at much longer intervals than prior to the institution of the treatment. There have been no fainting spells for the past four months. She occasionally has some trouble with deglutition, but does not complain of food retention. The patient is still gaining in weight (121½ pounds). Fluoroscopic examination of

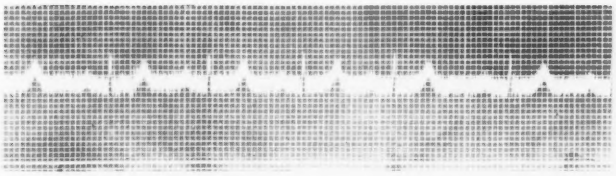

Fig. 2. (Before partaking of food) Lead 1. Normal conduction time.

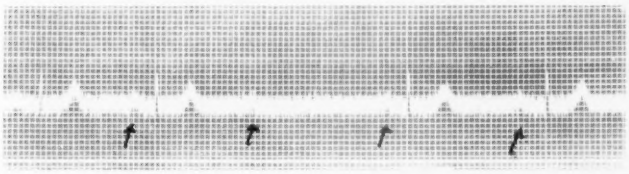

Fig. 3. Lead 1. Showing 2:1 block induced by swallowing food.

Fig. 4. Lead 1. Recurrence of transitory auricular ventricular block 2:1. (Several minutes after Fig. 3 was taken.)

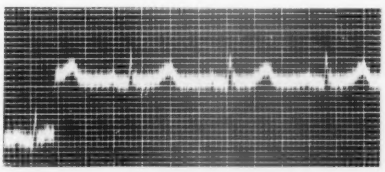

Fig. 5. Lead 1. (Taken while swallowing food.) Several months after repeated bouginage of the cardiospasm. Normal conduction time.

the esophagus still shows some spasm of the cardia, with delayed emptying time. The dilatation of the esophagus appears to be somewhat diminished. A recent electrocardiogram (April 11, 1936) shows no disturbance of the conduction apparatus during or after eating (Fig. 5). The patient continues to use the Hurst bougie at regular intervals.

COMMENT

The case just reported evidently represents a true case of transient complete heart block of reflex vagus origin. The absence of any signs of organic heart disease and the intimate association of the heart

block with the act of deglutition all point to a true vagovagal reflex originating in the dilated esophagus. The x-ray evidence of cardiospasm is manifest. An esophagoscopic examination (to which the patient has not consented) may throw some further light on the condition of the esophagus. Under bouginage treatment the patient shows marked improvement, but still has occasional signs of cardiac and esophageal disturbance. When the patient consents we intend to divulse the cardia with a hydrostatic dilator, and hope thereby to break the vicious circle. This procedure can be safely carried out, provided a preliminary dose of atropin is administered.

In view of the fact that cardiospasm is a disease of relatively frequent occurrence, and considering the intimate relation of the vagus nerve to the esophagus, it is rather remarkable that similar cases of intermittent heart block have not been reported as a complication. Further investigation may show that some of the symptoms usually described under cardiospasm may really be due to some hitherto unrecognized reflex interference with the cardiac neuromechanism.

SUMMARY

A typical case of cardiospasm has been described. When the patient partakes of food, syncope occasionally ensues, during which transient complete heart block can be demonstrated in the electrocardiogram. Between attacks the heart action is normal. The case is regarded as a typical one of vagovagal syncope. An attempt has been made to interrupt the reflex arc by dilating the cardiospasm, and the patient's condition is much improved.

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LXX

BRONCHOSCOPIC OBSERVATIONS IN ASTHMATIC CHILDREN*

EMILY LOIS VANLOON, M.D.

PHILADELPHIA

One patient who shows dramatic improvement after treatment will focus our enthusiasm on that treatment far more forcefully than many routine cases which respond mildly to our therapeutic efforts. Such a case was eight-year-old Tommy, who came to my office in October, 1934, with such urgent dyspnea that at first glance I thought of tracheotomy. But mirror examination revealed a normal larynx, and history suggested asthma. Tommy had been having severe asthmatic attacks since he was three years old. His previous treatment had included elimination of foods and other substances to which he was allergic and the administration of medication, including ephedrin. When I first saw him he was very much underweight, weighing 53½ pounds, which was at least ten pounds less than his twin sister. The present attack lasted for almost two weeks and the child presented a picture of exhaustion. He was sent to the hospital immediately and bronchoscopy was done the next day. We found a large quantity of viscid, purulent secretion blocking his airway, and after aspiration of this noted that his dyspnea had been still further increased by the tremendous accentuation of the normal expiratory anteroposterior bronchial narrowing. After bronchoscopic removal of the secretion, the dyspnea was relieved and the child went to sleep peacefully for the first time in two weeks. His further progress was spectacular. At varying intervals, four more bronchoscopies were done for removal of secretion. A vaccine was prepared from this secretion and administered in small doses over a prolonged period. He was referred to Dr. Jean Crump, who has worked with me in the care of these asthmatic cases, and further treatment, including desensitization with house dust and ragweed pollen, was given. Tommy now weighs 80 pounds, a gain of 27 pounds in 18 months; he has had no asthma since this treatment was begun except one mild attack during the ragweed season.

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*Presented before the nineteenth annual meeting of the American Bronchoscopic Society, Detroit, May 27, 1936.

In the last two years we have examined and treated bronchoscopically eighteen asthmatic children, ranging from three to twelve years of age, and we believe that endoscopy gives much useful information. Only by bronchoscopy can we be sure that the asthmatic wheeze is not due to a foreign body; Dr. Jackson, in 1931, reported thirty-seven cases of foreign bodies in the air and food passages of children in whom the condition had been erroneously diagnosed as asthma.

Again, we can obtain secretion for the preparation of vaccine. Allergists now concede that, particularly in children, infection plays an important rôle in precipitating asthmatic attacks in the susceptible individual. In the cases here summarized we find in the history "attacks began after measles," or "after pneumonia," "after severe bronchitis," "after persistent head cold," in almost every case. Therefore, the rationale of autogenous vaccine therapy in these children is clear; whether they are allergic to their own organisms, or infection increases their sensitivity to other allergens, immunity conferred by vaccines should lessen the frequency of attacks. We tried to compare the bacteriology of the bronchoscopic secretion with that of the patient's sputum, but in most of the cases it was impossible to obtain sputum. Sometimes the secretion was so scanty or viscid it could not be coughed up or the child was too young to expectorate the material. In the cases where sputum could be obtained, we found organisms in the bronchoscopic secretion (once hemolytic streptococcus) which were not present in the sputum.

In the culture of the bronchial secretion, we found nonhemolytic streptococcus 13 times; m. catarrhalis, 12; non-hemolytic staphylococcus, 8; hemolytic streptococcus, 7; pneumococcus, 5, and influenza once.

These children almost always have nasal symptoms which sometimes appear to be vasomotor only, but more often suggest definite purulent sinusitis. Clinical and roentgen ray examination revealed sinusitis in 65 per cent, and the common history of frequent colds, which often usher in severe asthmatic attacks, can not be ignored. Appropriate treatment to the nasal infection is of paramount importance. We have taken nasal cultures from these little patients and find that they in no case correspond accurately with the bronchoscopic culture. Repeatedly we find streptococcus in the bronchoscopically removed secretion when it is not present in the nasal cultures. This discrepancy strongly suggests that the use of vaccine prepared from the bronchoscopic secretion can not be well replaced by vaccine made from the more easily obtained nasal cultures.

The reports on x-ray films of the chests of these patients frequently include the statement "there is increase in the hilar and bronchial markings suggestive of bronchiectasis." With the increased pressure from coughing and the expiratory dyspnea, bronchiectasis might well be expected. Even more might this be expected when we consider how frequently there is present that other factor often associated with bronchiectasis, namely sinusitis. In four cases where the film was suggestive we instilled lipiodol and found no bronchial dilatations. This number is too small to be conclusive, of course, and further bronchographies will be done to determine the presence or absence of bronchiectasis in asthmatic children.

We have recorded our bronchoscopic findings in these children, noting the character and amount of the secretion, the color of the membrane, the amount of anteroposterior collapse, and the relationship of these factors to the history and therapeutic result. Unfortunately these findings show no uniformity. All but three cases had varying degrees of congestion of the mucous membrane; in these three the membrane was pale. One of these three was the case previously reported, with splendid results from treatment. One was a child who is allergic to feathers, wool and many other substances and was apparently somewhat better after vaccine therapy, and one was in a patient who was unimproved after bronchoscopy and administration of vaccine. Generally speaking, in the patients whose congested tracheobronchial mucosa suggested the presence of infection, the results of vaccine treatment are most satisfactory.

The secretion was scanty in amount in ten cases, moderate in six and plentiful in two. Where only a few drops of secretion were encountered, it was usually viscid and appeared in a lump, clinging to the bronchial walls; the more profuse secretion was usually very thick and frankly purulent. We examined some of the specimens for eosinophiles and found eight to ten per L.P. field in some cases and none in others who were just as definitely allergic.

The presence of bronchospasm in asthma has been widely discussed in the literature. We saw nothing suggestive of spasm, but an accentuation of the narrowing of the bronchial lumen on cough was an almost constant occurrence. This has been mentioned by Clerf and Lukens, who describe it as collapse. In the more marked cases the posterior wall almost meets the anterior wall on expiration or cough. It creates the impression, not of an active spasm, but of a lack of rigidity in the bronchial walls, a passive rather than an active

factor. We hoped that blood calcium would be low in the patients who demonstrated this most clearly, but this was not the case; the blood calcium findings and the degree of collapse could not be correlated. This anteroposterior collapse can well explain mechanically the expiratory dyspnea from which asthmatics suffer, for the lumen is sometimes diminished on expiration to a scabbard shaped slit through which little air can escape.

The most interesting part of this study, certainly the most interesting to the patient, was the result of treatment. The immediate result was almost always favorable. Particularly in the patients who had been having a prolonged asthmatic attack, immediate relief from symptoms followed, and the patient was able to get some much-needed sleep. Looking over the reports of nurses, one is struck by the frequency of the notation, "patient returned from bronchoscopic clinic; breathing more quietly." This improvement sometimes is permanent and sometimes lasts only a day. The ultimate results of bronchoscopy and the administration of vaccine prepared from the secretion so removed are generally very good. Of this series only one reported no improvement; one patient who received no treatment except vaccine has had no asthma since its administration eighteen months ago. One who had had asthma almost constantly for four years has had no attack since vaccine therapy was started. In some of the children, house dust and pollen desensitization were combined with the vaccine; in others, no other source of irritation was found and the vaccine alone was used. Bronchoscopy was repeated in occasional cases when thick secretion blocks the airway; this usually occurred after an acute rhinitis and bronchitis.

Summarizing the results, one has been unimproved, four have been moderately improved, four have been almost cured, having had only an occasional mild attack since treatment was begun, and in seven patients there has been symptomatic cure, with no recurrence of the asthmatic attacks. In two patients the time elapsed since bronchoscopy has been too short to permit a reliable report.

The question naturally arises as to whether the house dust desensitization and other treatment have not cured the cases in which it was used, rather than vaccine. Dr. Crump, who has followed her cases closely in a large asthma clinic at a children's hospital, definitely believes that the bronchoscopic vaccine has proved an invaluable adjunct to the other treatment. The children receiving it are less susceptible to attacks than those whose treatment does not include vaccine.

CONCLUSIONS

Bronchoscopy has proved itself of service in asthmatic children:

a. In differentiating foreign body or other bronchial obstruction from true asthma.

b. In relieving the dyspnea of acute attacks by removal of obstructing secretion; and,

c. By yielding secretion for the preparation of vaccine.

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Clinical Notes.

LXXI

ORBITO-ETHMOIDAL OSTEOMA, WITH A REPORT OF THE CASE*

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Osteomata of the nasal accessory sinuses are uncommon. This is demonstrated by the limited number on record, and the rarity with which more than two are reported by any one author. Of those recorded, by far the greatest number were located in the frontal sinus. A recent tabulation by Carmody,[1] of 139 cases, lists only eight in the ethmoid labyrinth. Apparently an osteoma arising in and limiting its growth to the ethmoid is rather rare.

Orbito-ethmoidal osteomata may arise from any portion of the ethmoid labyrinth, but the commonest site is at the junction of the orbital plate and the ethmoidal roof.

The etiology of these tumors is debatable. Cloquet believed them to be ossified polyps. Virchow thought they sprang from the diploe of the skull, and referred to them as enostoses. Arnold's theory was based on developmental cartilaginous rests. Fetissof,[2] confirming Pfeiffer's and Eckert's investigations, believes that they originate from the periosteum.

Cushing[3] stresses trauma as a factor in stimulating "bone production activity." Eagleton[4] found infection present in all of his cases and argues that it is "chronic bacterial irritation applied to an area of osteogenic bone" that produces the growth.

There are three types, namely, eburnated, spongiose and mixed, the latter predominating. They are usually attached by a cancellous pedicle to their base.

Fetissof states that the fibrous tissue filling the interstices of the spongiose bone is a direct extension of the periosteum covering the

*Presented before the meeting of the Western Section of the American Laryngological, Rhinological and Otological Society, Del Monte, Cal., February 2, 1936.

tumor, and that its growth is from within outward by metaplasia of fibrous tissue into bony tissue, the peripheral ossification denoting a secondary rôle. He admits that it is difficult to differentiate histopathologically between osteomata and exostoses, but insists that this must be done clinically to insure a surgical cure. Macroscopically, according to Fetissof, an exostosis is the result of diffuse hyperplastic bony changes, probably of an inflammatory nature, while an osteoma is a neoplasm capable of unlimited growth, with displacement of adjacent structures. Surgical removal of the excess bony tissue of an exostosis is sufficient, while with the osteoma, its pedicle as well as the base from which it arises must be removed to prevent recurrence.

Cases are on record of the growth lying free in the sinus with the pedicle detached, and these are spoken of as dead osteomata. Sewall[5] found no attachment to the sinus wall in his case.

Most writers agree that the tumor's ultimate extension and destruction is unpredictable and early removal is indicated. A review of Cushing's four cases with intracranial extensions should end vacillation.

REPORT OF THE CASE.

CASE 1.—Orbito-ethmoidal osteoma with moderate exophthalmos of six weeks' duration. Removal of the tumor through a fronto-ethmoidal incision. Attached to the roof of the anterior ethmoidal labyrinth, just posterior and internal to the suture between the orbital plate of the frontal bone and the adjacent edge of the ethmoidal roof. Eburnated type.

R. C., male, age 18 years. First seen by Dr. R. J. Sprowl, September 9, 1935. Chief complaint, moderate protrusion of left eye. Patient's mother states that it had been evident for six weeks and had increased noticeably during the past three weeks.

The antecedent history had no apparent bearing on his present trouble. He had been playing football, but there was no history of specific frontal injury. There had been recurrent epistaxis for years. Mumps, two months previous.

There had been some blurring and tearing following use of the eyes for several months, and occasionally a mild, left frontal headache.

Eye examination: Unilateral exophthalmos. Left eye displaced downward and outward. Rotation limited nasally, with diplopia elicited in the right temporal field. Left upper lid moderately swollen, but no inflammatory changes. Media clear throughout. Fundus negative. Fields and blind spot normal. V O U 20/20.

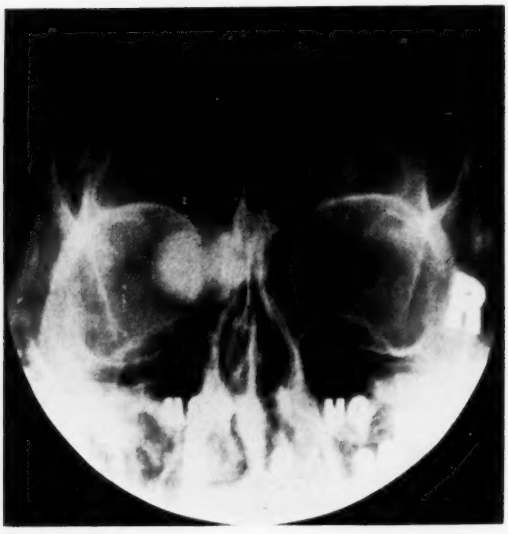

Fig. 1. Radiogram of tumor. Anteroposterior view.

Fig. 2. Radiogram of tumor. Lateral view.

Fig. 3. Photograph of tumor. *X* marks pedicle which was attached to dural plate.

Nasal examination: There was marked high deflection of the nasal septum to the left, which obscured a clear view of the ethmoid area. The bulla was enlarged. The mucosa was normal in appearance and no pus could be seen. A superficial enlargement of the veins in Kiesselbach's area on each side of the septum accounted for his recurrent epistaxis.

X-ray report by Dr. Milo Harris: "An osteoma apparently arising in the roof and medial wall of the left orbit and extending into the orbit and the ethmoid cells. The sinuses appear clear."

September 18th, operation under local anesthesia. Fronto-ethmoidal incision. A very large frontal sinus encountered with normal mucosa. After opening two anterior ethmoid cells a white, glistening tumor of ivory hardness was encountered. It extended to the septum internally and well into the orbit externally. The orbital plate was absent. The growth was covered with a congested, moderately thickened mucosa which stripped off easily. It was attached by a pedicle to the roof of the ethmoid labyrinth. The tumor was grasped with a large forcep and with a gentle rocking movement was freed and delivered in one piece. The pedicle fractured clean from its attachment and the dural plate remained intact. A few remaining ethmoid cells were exenterated and a very thin shell of the middle turbinate removed. The external wound was closed without drainage.

On the third day some puffiness of the upper angle of the wound appeared and a small amount of serum was evacuated. This mild drainage continued for several days. Diplopia was present for three weeks. The exophthalmos disappeared slowly and when last examined the eye was normal in position, rotation and vision.

PATHOLOGICAL REPORT BY DR. J. D. EDGAR.—The specimen consists of a hard irregular bony mass weighing 28.0 gms. Its maximum dimensions are 3.6 x 3.8 x 1.9 cm. It is made up of a cylindrical, slightly bosselated, whitish basal portion with a short stout pedicle near one end, by which the mass was attached to the ethmoid roof. The point of attachment measures 0.6 x 0.4 cm. and the pedicle is 0.8 cm. long. From the other end of the basal portion springs a large protuberance resembling the half of a doughnut which straddles the basal part and is inclined slightly forward. This large knob is smooth and yellowish. Grossly no covering membrane can be found. On sawing through the specimen it is found to be brittle and made up of very dense eburnated bone. The surface is homogeneous, all parts of the mass having the same gross structure.

Microscopic sections of the decalcified tumor mass stained with hematoxylin and eosin and Van Gieson's stain show the superficial portions composed of dense bone. The pattern is distorted. Regular Haversian systems are lacking, the lacunæ being irregularly arranged. Strands of fibrous tissue which take a paler stain interlace between the calcified trabeculæ. This fibrous tissue is poorly demarcated, the peripheral fibers fading imperceptibly into the bone. In the central portion of the specimen the bone is spongiose, the bony trabeculæ being relatively massive. Here the fibrous tissue has disappeared. Throughout the tumor the bony architecture is distorted. The lacunæ show a definite tendency to be arranged in rows parallel to the fibrous bundles.

Pathologic diagnosis: Benign eburnated osteoma of the ethmoid sinus.

COMMENT.

The microscopic structure suggests an origin from fibrous processes originating in the periosteum.

PAULSEN MEDICAL AND DENTAL BLDG.

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LXXII

ABERRANT VESSEL OF THE POSTERIOR PHARYNGEAL WALL.*

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Boston.

Massive pharyngeal hemorrhage is not only alarming but frequently fatal. Hemorrhage following the incision of retropharyngeal and peritonsillar abscess occurs with sufficient frequency that it should be considered as a possible complication. Visible pulsating pharyngeal vessels are not uncommon. Carmack's thesis for this Society was entitled "Aberrant Internal Carotid Arteries," and more recently MacCready discusses the "So-called Aneurysms of the Ascending Pharyngeal Artery."

REPORT OF A CASE.

History.—A child, four years of age, was admitted to the Massachusetts Eye and Ear Infirmary, September 28, 1935, on account of a retropharyngeal swelling that was causing respiratory difficulty. There was no temperature or pain; the blood count was normal. The swelling occurred fairly rapidly until it filled the pharynx and extended into the postnasal space. It did not pulsate, was definitely nonexpansile, nonfluctuating and appeared to be encapsulated.

Past History.—Four months ago this child had been a patient in this hospital on account of hemorrhage following the incision of a retropharyngeal abscess by a competent laryngologist. During her two weeks' stay in the hospital two transfusions were necessary. Since her discharge she had been seen in the Out-Patient Department regularly and the rapid swelling on the left side of the pharynx developed under observation

Comment.—In making a differential diagnosis, cyst, tumor and aneurysm had to be considered. The past history of an incision of a retropharyngeal abscess followed by a hemorrhage that necessitated transfusion suggested the possibility of an injury to a pharyngeal vessel. The fact that the child survived made it unlikely that this vessel was an abnormal internal carotid artery. Tumors in children are not uncommon, and cyst of the pharynx had to be considered.

*Read before the Eastern Section of the Laryngological, Rhinological and Otological Society, Newark, N. J., January 3, 1936.

Operation.—On November 1st, under gas-ether anesthesia, with the patient in the Rose position and a Sewall-Davis mouth gag in place, the soft palate was retracted by Beck's method, giving an excellent exposure of the swelling. An aspirating needle was inserted into the mass in several places, but I was unable to aspirate either cystic fluid or blood.

An incision through the mucous membrane one and a half inches in length was made over the point of maximal swelling. The mucous membrane was dissected laterally on each side of the incision for a distance of one centimeter. This dissection exposed a firm, adherent fibrous layer. Again the aspirating needle was inserted and again no cystic fluid or blood was obtained. An incision was made through the firm, fibrous layer, and through this incision old clotted blood exuded. As this blood clot welled up through the incision, it was removed by suction and then a large blood vessel mounded up in the incision. The vessel wall appeared intact, and as there was no bleeding the incision was closed with interrupted catgut sutures. Examination now showed that the mass had almost completely disappeared. I felt that I had evacuated a hematoma caused by an injury to a pharyngeal vessel.

On the evening of the third postoperative day there was a severe hemorrhage from the nose and mouth that was controlled by postnasal packing. The patient's blood was grouped for transfusion, a donor obtained and kept available. Her blood count was 3,300,000 with a hemoglobin of 80 per cent. In anticipation of possible ligation of the carotid, the artery was compressed against the vertebræ for five minutes every hour. The postnasal packing was removed on the morning of the fourth postoperative day.

That evening, while the child was sitting up in bed, she suddenly had a very violent hemorrhage from the mouth, losing approximately ten ounces of bright red blood within ten seconds. The child dropped as if struck and seemed pulseless. The common carotid artery was immediately compressed against the vertebræ and this somewhat controlled the violence of the hemorrhage. Suction and all equipment were fortunately at hand. A large sponge was jammed into the pharynx over the incision. By the use of suction the bleeding was seen to come from the upper end of the incision. A tonsil sponge in a curved clamp, pressed firmly against this bleeding point in conjunction with pressure on the carotid, stopped the bleeding. The child was extremely pale and almost pulseless. When I arrived, twenty-five minutes later, I found one house officer in sterile cap, gown and gloves holding this sponge in the nasopharynx with one hand

and compressing the carotid artery with the other. Another house officer was taking blood from the donor for a transfusion. The child had been draped for operation and the neck prepared and anesthetized with novocain. While I tied the common carotid artery the child received a transfusion of 300 cc. of blood. Tying the common carotid artery completely stopped the bleeding from the throat. The child was in profound shock, and shock measures were adopted so that within a half hour the blood pressure reached 100. An intravenous injection of saline was continued for four hours and the next day the blood count was 4,500,000.

Following the operation there was no further bleeding. The tumor mass had almost completely disappeared and the patient suffered no neurologic or cerebral symptoms.

In retrospect, my title may not be absolutely correct, but this case of massive hemorrhage is reported as a tribute to my house officers who were prepared for the complications that occurred.

I wish to acknowledge my indebtedness to Doctors Mertens, Lentine, Lynch and Brown for their handling of this patient.

270 COMMONWEALTH AVE.

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LXXIII

PRIMARY ADENOCYSTIC CARCINOMA OR CYLINDROMA OF THE TRACHEA.

M. Reese Guttman, M.D.,

Chicago.

The relative rarity of primary malignant disease of the lower portion of the trachea is a matter of common knowledge. In fact, Simpson and Moore[1] were able to find but twenty-seven cases, which included one of their own, in their extensive review of the literature on the subject. In but twenty of the reported cases were the histologic descriptions of the neoplasms sufficiently clear so as to permit of accurate classification. Eleven of the series were glandular in type, cylindrical cell or adenocarcinoma. The high incidence of this type of malignant growth can be anticipated in view of the histologic structure of the tracheal mucosa with its stratified columnar epithelium and the profusion of mucous glands found within it. Eight of the series were typical squamous cell carcinomata. The origin of squamous carcinoma is a bit difficult to envision as arising from the epithelium of the mucous glands or the cylindrical cells of the tracheal mucosa. Metaplasia may account for this phenomena, but in all probability these types of growth arise from cell nests of the primary embryonic epithelium of the primitive foregut from which the respiratory system develops. D'Aunoy and Zoeller[2] are especially inclined to this latter view. Only one was a basal cell carcinoma arising, no doubt, from the basal layers of the tracheal mucosa. We have recently encountered another case of primary carcinoma of the trachea which might be of interest, not only from the point of view of the reported rarity of these lesions but also on account of the histologic features that were exhibited, and not as yet having been found in the trachea. Adenocystic carcinoma of the trachea has not been previously described in the literature.

REPORT OF A CASE.

Case 1.—Mrs. I. D., age thirty-two, was seen on October 12, 1934, with the complaint of marked difficulty in breathing. Her difficulty began about two years prior to the date of her consultation. Previous to that time she had been in fairly good health. She gave a history of an appendiceal operation and also had a gynecologic con-

dition that was improved by an abdominal procedure. Some time in 1932 she contracted what was diagnosed as asthma. Her main complaint at that time was irregular attacks of wheezing, which came on after physical exertion. As time progressed her wheezing became more pronounced, and finally she began to notice that her breathing became more difficult and "puffy" after exertion. Within the past six months her difficulty in breathing has been so pronounced that physical effort is almost impossible. She also noticed that after falling asleep she would be awakened by a strangling sensation and find herself struggling for air. Within the past month this strangling sensation and the associated struggling for air would come on whenever she exerted herself and consequently she has been forced to remain quiet, usually in a sitting position.

Examination disclosed an adult half breed Indian female, highly nervous and speaking in an aphonic whisper. She was decidedly dyspneic, having a pronounced crowing inspiratory stridor. She was unable to talk above a whisper. Speech was difficult and only a few phrases could be uttered without pausing. Her nose disclosed a slight deflection of the septum to the right. Her teeth were in excellent condition. The pharynx was negative. The tongue showed some hyperplasia of the lingual lymphoid mass. The larynx disclosed a picture of injection throughout. The cords moved freely on attempted phonation and inspiration. Her chest examination disclosed nothing of importance, except for a marked inspiratory and expiratory wheeze heard throughout, but most marked over the trachea. Her abdomen was negative except for a midline and right rectus incision. Her extremities and reflexes were negative. On account of her urgent dyspnea and her state of exhaustion an immediate tracheotomy was decided upon.

Operation was performed under local anesthesia. It progressed without unusual incident until the trachea was laid bare and ready for incision. As a preliminary to opening the trachea it has been our custom to inject a few drops of cocain into the tracheal lumen in order to prevent the excessive cough so frequently encountered on opening the trachea. It was surprising to note that the hypodermic needle did not enter an open space upon penetration of several millimeters. The trachea was incised but instead of the expected lumen a red firm mass was encountered. The mass was incised cautiously for about five millimeters when the tracheal lumen was encountered. A short tracheal cannula was inserted but did not prove adequate, as it did not reach beyond the growth. A Jackson cane cannula was obtained, inserted and found to be satisfactory. A portion of the

tumor was excised and sent to the laboratory for examination. The wound was closed in the usual manner. The patient experienced immediate relief from her dyspnea. In spite of the presence of the tracheal tube, to which her trachea had not become tolerant, she was able to sleep the entire night.

Histopathology.—The sections were stained with hematoxylin and eosin. The tumor consisted of nests of epithelial cells forming islands or acini. The cell nests varied in size and shape, with a tendency to being roughly ovoid, although many were decidedly irregular in outline. The nests were surrounded by a connective tissue stroma. The stroma was made of a rather vascular connective tissue. The islands and acini were formed of compactly arranged columnar epithelial cells. The cytoplasm of the cells was eosinophilic and the nuclei oval in shape. The cells varied but little in morphology and resembled the basal cells of the epidermis. Mitotic figures were infrequently seen. In the lumina of some of the cell groups is found an amorphous basophilic material. The diagnosis by our pathologist, Dr. Otto Saphir, was "Epithelioma adenoides cysticum (variety of basal cell carcinoma)."

Bronchoscopy.—Endoscopic examination was made several days later. The tracheal mucosa on the right side was found to be elevated and infiltrated and dark red in color. The infiltration extended from the subglottic region above down to within two centimeters of the origin of the right main bronchus. The mass occupied the right side of the tracheal circumference, passing beyond the midline anteriorly to invade three to five millimeters of the left side. Posteriorly it did not quite reach the midline of the tracheo-esophageal party wall. The mucosa overlying the infiltrate was red and congested but showed no erosion of the underlying tumor at any place. The remaining mucosa of the uninvolved portion of the trachea was but slightly reddened. The tracheal lumen showed a crescentric, irregular constriction on the right side. Palpation of the growth with the tip of the bronchoscope gave one the sensation of firm tissue almost cartilaginous in texture.

Roentgenology (Dr. Sidney Mesirow).—Lateral radiologic examination of the neck reveals a stenosis of the tracheal lumen below the level of the cricoid cartilage, extending downward for about two inches, caused by an apparent bulge exteriorly into the lumen. The lesion is apparently posterior and unilateral. Whether this is the result of an intrinsic tumor of the trachea, or a mass projecting from behind and pushing the tracheal lumen before it, is not definitely

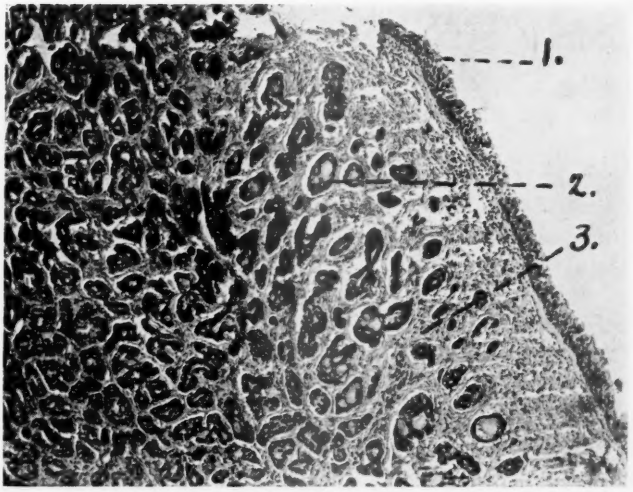

Fig. 1. Microscopic structure of adenocystic carcinoma of the trachea. *1.* Tracheal mucosa. 2. Cell nest. 3. Connective tissue stroma.

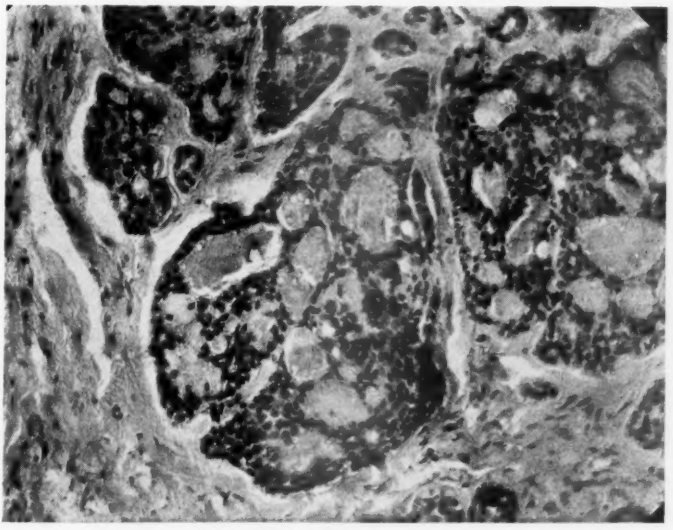

Fig. 2. Higher magnification to show detailed structure of the cell nests and their contents.

determined. The laryngeal ventricle is not visualized, but this may be the result of lack of air filling, rather than actual pathology. A tracheotomy tube is noted just above the sternoclavicular junction.

The patient recovered rapidly from her tracheotomy, but insisted upon leaving for her home town for further care. She was advised to have deep x-ray therapy, as the extension of the neoplasm to the region of the origin of the right main bronchus would preclude surgical removal of the trachea. When last heard from in September, 1935, she was still alive, although leading a cannula life.

DISCUSSION.

Adenocystic carcinoma of the mucosa of the respiratory passages has been but infrequently described. These lesions have been reported, especially in the foreign literature, as cylindromas, basal cell carcinomas and certain types of endotheliomas. In consequence of the use of these various terms in describing histologically comparable lesions, confusion has arisen in the classification. Bredlau,[3] in a most comprehensive review of the subject, recommends that they be recognized as varieties of basal cell carcinoma and should be so classified, with the addition of the descriptive terms of solid, cystic, adenoid or adenocystic, as was advocated by Krompecher.[4]

Clinically the characteristic feature of these neoplasms is a mildly malignant tumor that grows slowly, tending to progress by submucous prolongation penetrating the overlying mucosa or the surrounding tissues late, if at all. Recurrence is the rule after local removal, although it may be delayed for months and even years. Metastases but infrequently occur, the constant local recurrence and local extension being the most important evidence of malignancy.

Histologically the neoplasms are characterized by nests of slightly anaplastic epithelial cells, closely resembling the basal cells of the surface epithelium. The cell nests are commonly presented as anastomosing cords. The nests may vary in size and shape, appearing round, ovoid or irregular. Most nests have an intact marginal layer of cells which vary from polyhedral to columnar in shape. The cell cytoplasm is small in amount and homogeneous in staining. The nuclei are rounded or ovoid and are finely granular. Many of the nests and cords may exhibit a lumen that may contain desquamated cells, granular or fibrillar débris or a homogeneously staining hyaline like material, giving the nest a cystlike appearance. The nests and cords are united by a connective tissue stroma, which may be loose or dense, and is frequently highly vascular. Not uncommonly, few or no cell

nests with a lumen may be seen. This gives the characteristic picture of the well known basal cell carcinoma.

The classification and histogenesis of these tumors have been in dispute for some time, and have been the cause of the confusing nomenclature. In Bilroth's[5] original contribution he described a tumor of the orbit that recurred seven times and did not metastasize. The tumor was characterized by cell nests that were surrounded by a definite hyaline ring lying between the stroma and the tumor cells. He coined the term of cylindroma for this lesion, which he believed to be of epithelial origin. Volkmann[6] and Citelli[7] described cylindromas as of endothelial origin, while Kolaczek[8] considered them to be angiosarcomata. Pfeiffer[9] reported two cases of cylindroma with characteristic cell nests and anastomosing cords and assumed that they were of endothelial origin. Barbezet[10] described a case with characteristic histology and concluded that they were of epithelial origin, probably from the nasal mucosa. It remained for Krompecher[4] to clarify the subject in his extensive and detailed consideration of these neoplasms. He pointed out that basal cell carcinomata of the skin and mucous membranes are very similar histologically, and in many instances difficult or even impossible to tell from the microscopic appearance whether the basal cell growth originated in the skin or mucous membrane. He was able to trace their origin from the basal layers of the skin or mucous membrane or of the basal layers of the various structures found in them, such as hair follicles, sebaceous, serous and mucous glands. He described the commonly accepted picture of basal cell carcinoma of skin consisting of solid masses of cells and the not infrequent appearance of cell nests and cords, many exhibiting a hyaline-containing lumen in these lesions of the skin. He further showed that the characteristic lesion of a basal cell growth of mucous membrane was of the adenoid and cystic types, although occasionally a picture of solid cell mass was presented similar to that found in basal cell carcinoma of the skin. He thus stressed the fact that these lesions were all basal cell in origin and should be classified as such. He further indicated that they may occur as solid adenoid or cystic types, the solid type being characteristic of those dermal in origin and the adenoid and cystic types characteristic of these of mucosal derivation. He further concluded that the clinical course of these tumors was similar, namely, relatively benign, with marked tendency to a long course over a period of time reckoned in years and characterized by a slow, progressive tendency to infiltrate submucously and not metastasize and recurring after local removal. Ewing[11] is in accord with Kompecher in considering these neoplasms

as basal cell carcinomata, as does Davies[12] and Stillians.[13] These tumors have been described as presenting on the skin, scalp, orbit, nose, paranasal sinuses, salivary glands, palate and tongue. But one reported by Eigler[14] was located in the larynx and none in the trachea.

SUMMARY.

A rare type of basal cell malignancy (adenocystic carcinoma or cylindroma) is reported as a primary growth in the trachea, bringing the total recorded cases of primary malignancy of this organ to twenty-eight. Histologically, these neoplasms exhibit anastomosing cords and nests of slightly anaplastic cells that closely resemble basal cells of surface epithelium. The cell nests present a central lumen that may contain cellular débris or a hyaline-like material.

Grossly the neoplasm tends to grow slowly and infiltrate its way beneath the tracheal mucosa. The lumen of the trachea is irregularly eccentrically reduced. The overlying mucosa appears red and congested. There is no tendency for the neoplasm to erode the mucosa or to metastasize to regional lymph glands or the abdominal viscera.

Characteristic symptomatology is the progressive increase in dyspnea, associated with otherwise apparent good health.

Characteristic roentgen finding is the irregular constriction of the tracheal lumen by a submucous infiltrate.

Bronchoscopy reveals a smooth, irregular constriction of the tracheal lumen, due to a submucous infiltrate that feels firm to the exploring tip of the bronchoscope. The overlying mucosa is red and congested, while the noninvolved portion of the tracheal mucous membrane may be normal in appearance.

Diagnosis can only be definitely established by biopsy.

Treatment consists in cautious administration of deep x-rays with due regard for the tracheal cartilageous.

Tracheotomy is indicated for the dyspnea.

The prognosis is doubtful as to ultimate cure, although the life expectancy can be reckoned in years.

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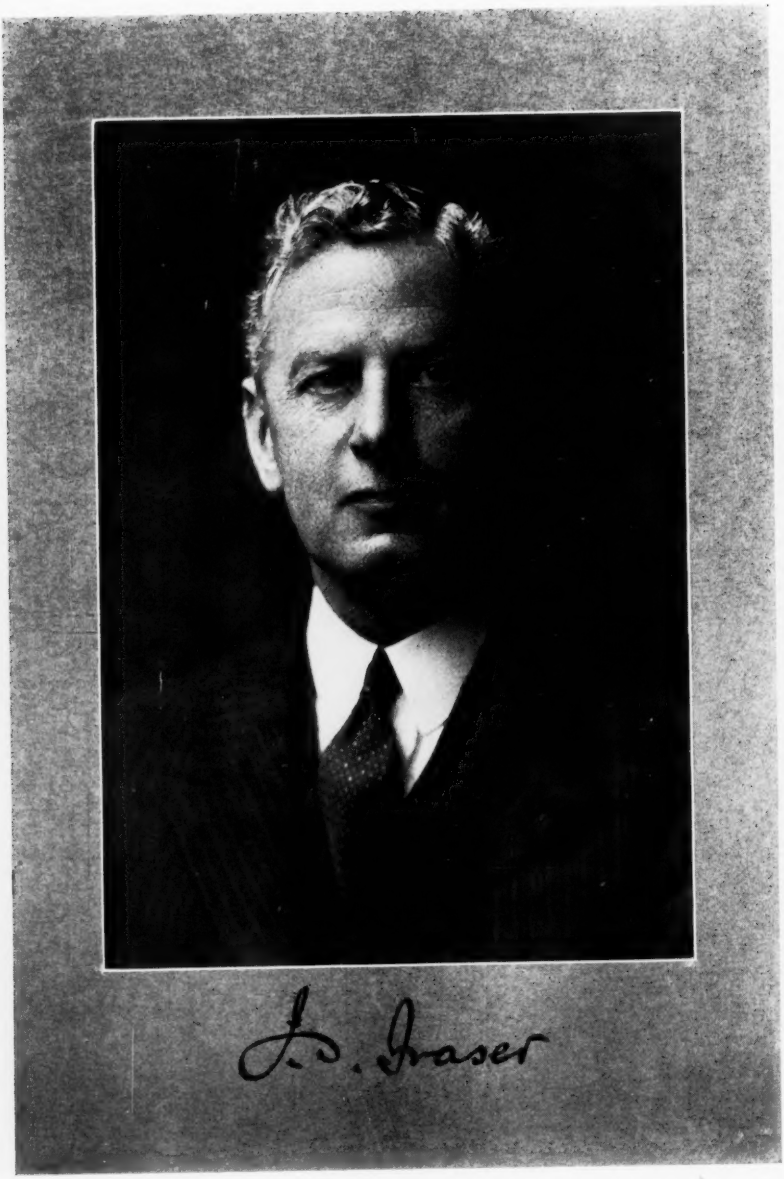
L. Fraser

J. S. FRASER

1874-1936

British oto-rhino-laryngology has lost in the last eight months no less than eight personalities. The most recent of these is J. S. Fraser, who died on May 11th in Edinburgh.

Fraser was born in 1874 in Fife and went to Edinburgh at the age of six. He qualified M.B., Ch.B. in 1897, with first-class honors. As a foundation for his career he acted as an Assistant in a general practice in Lichfield for five years.

In 1904 he began his work in ear and throat and studied in London and Vienna; he then returned to Edinburgh as Assistant in the Ear and Throat Department of the Royal Infirmary. In 1905 he became a Fellow of the Royal College of Surgeons of Edinburgh and the next year was appointed to the Staff of the Infirmary and was Senior Surgeon in the Ear and Throat Department at the time of his death.

Fraser was one of those who maintain a youthful appearance; his youth was not only in his face but in his outlook on life and work: keenness was his characteristic; he was an energetic worker in every direction; socially, clinically, in the operating theater and last but far from least, research: indeed it may be truly said that he was one of the world's outstanding research workers in otology: his microscopical research was in the forefront of all such investigations.

Fraser's papers at the Otological Section of the Royal Society of Medicine were delightfully lucid and he held his audiences as few others could. His papers were on a wide range of subjects and were often illustrated by microscopical sections of the temporal bone all cut by himself; they demonstrated the changes found in congenital syphilitic deafness, otosclerosis, deafmutism, suppuration in the internal ear.

It is not surprising that all this pathological research placed him very high in the otological world. He was an honorary member of the Austrian and American Otological Societies and of the Otological Society of Madrid and a corresponding Member of the American Laryngological Association. He was President of the Otological Section of the Royal Society of Medicine in 1934: and at various times he was the recipient of all the important prizes for otologists.

Fraser was an admirable organizer; this was amply demonstrated by the large amount of work that emanated from his Clinic; statistical figures on many branches of otology published by him and his assistants were models of honest detail.

He wrote as lucidly as he spoke and his article in the Edinburgh Text Book on Ear, Nose and Throat, edited by Logan Turner, is the best short account of the ear and its diseases in the English language: he was the obvious man to represent British Otology in the discussion on otosclerosis at the International Congress at Madrid.

Fraser was a most charming companion, always cheerful and a delightful conversationalist ready to discuss any subject, professional or otherwise. His death leaves a large gap among his many friends and colleagues, not only in Edinburgh but wherever otologists foregather.

W. M. Mollison.

Abstracts of Current Articles.

NOSE

A Comparison of Intradermal and Intramucosal Tests in Cases of Allergic Rhinitis.

Linton, Lloyd D. (St. Louis), Arch. Otolaryng., 23:679 (June), 1936.

The reactions to intradermal tests are not a reliable index of the sensitivity of the nasal mucous membrane.

Seven patients had symptoms in the ears on entering the clinic; in six of them the symptoms were induced by the intramucosal injection of allergen in the nose.

Asthmatic attacks were induced by intramucosal tests in some asthmatic patients.

Headaches were produced in eight of the ten cases in which headache was an entrance complaint.

No one allergen was predominant in producing positive reactions when the symptoms produced were other than those of uncomplicated rhinitis.

The offending allergens can be determined in most persons with negative cutaneous reactions by intramucosal tests.

TOBEY.

LARYNX

Acute Laryngotracheitis in Children: A Study of Forty-three Cases Occuring at the Children's Hospital, Los Angeles, in Epidemic Form, During the Fall and Winter of 1933-1934.

Smith, W. Jewell (Phoenix, Ariz.), Arch. of Otolaryng., 23:420 (April), 1936.

Acute nondiphtheritic laryngotracheitis in infants and children may occur in epidemic form. The pathologic process is found principally in the subglottic tissues. This is because in the infant and the small child there is a predominance of loose areolar tissue that allows swelling to occur and shut off airway.

The findings in cases of severe involvement are those of air hunger, viz., retraction and restlessness. The condition calls for immediate action, and surgical intervention should not be delayed. Provision must be made for adequate postoperative treatment. The condition results in high mortality.

As most observers have found, the mortality from this condition is high. In this series of forty-three patients, twelve, or 27.9 per cent, required surgical intervention in the form of tracheotomy. Of the

twelve so treated, four died—a mortality of 33.3 per cent. Considering the entire series, there was a mortality of 9.3 per cent.

TOBEY.

ESOPHAGUS

Esophagitis: II. A Pathologic and Clinical Study.

Butt, Hugh R., and Vinson, Porter P. (Rochester, Minn.), Arch. Otolaryng., 23:550 (May), 1936.

It has been shown that because of its anatomic structure the esophagus is vulnerable to the organisms of the oral cavity, the regurgitated gastric contents and infection through lymphatic structures from the abdominal viscera.

Experimentally, it has been proved that the mucus which normally covers the lining of the tube is the greatest protective barrier. Lesions heal quickly in a normal esophagus, but without the protective mucus layer and with slight irritation the lesions go on to form ulcers. Microscopically one often observes that the mucous glands are blocked and dilated or have been destroyed by infection, hemorrhage or thrombosis. In this manner the supply of mucus is diminished. Rapid dilatation and strong movement of the tube, as a result of vomiting or stimulation of the central nervous system, removal of gastric contents by tube or infarcation are all capable of producing esophageal lesions. These lesions, with poor protection owing to diminished secretion of mucus, are constantly being irritated by normally regurgitated gastric content. Thus the inflammatory process is initiated.

In their series vomiting and the removal of gastric contents were the only factors which showed a relation to the increased incidence of esophagitis. However, infection, stimulation of the central nervous system and a poor blood supply are all factors which may initiate a lesion.

Postmortem gastric analysis shows that during the last few hours before death there are not sufficient irritative properties in the gastric content to produce the esophagitis seen at the autopsy table. Esophagitis, then, is not entirely an agonal condition.

However, vomiting, they believe, is the important factor in the etiology of esophagitis. It seems reasonable to suppose that recurrent attacks of esophagitis occur more frequently at sites where healing, with the production of fibrous tissue, has occurred. Since normal persons are constantly regurgitating gastric contents into the esophagus, it seems reasonable to suppose that during a lifetime there may

occur many unrecognized attacks of esophagitis. When healing occurs fibrous connective tissue forms. If this process occurs frequently enough a benign stricture may result.

In the 3,032 necropsies performed, the incidence of esophagitis was 7.02 per cent. In 75.2 per cent of the cases the lesions were found in the lower third of the esophagus. In 76.5 per cent of the 213 cases in which it was present the esophagitis was of the acute ulcerative type. Definite symptoms occurred in 10.3 per cent of the cases, and the most common symptom was substernal burning, which occurred in 53.2 per cent.

TOBEY.

EAR

Performance Characteristics of Electrical Hearing Aids for the Deaf.

Hartig, Henry E., and Newhart, Horace (Minneapolis), Arch. Otolaryng., 23: 617 (June), 1936.

Among the various hearing aids of different manufacture on the market having as components a carbon microphone, a carbon amplifier and an ear-fitting receiver, there are important differences in the amount of amplification produced and tremendous differences in the intelligibility ratings.

The differences in performance do not depend measurably on the observed nonuniformity of response. In consequence, furnishing frequency response curves to the otologist with each hearing aid would be of no value.

Of greater importance is a wide range of response (as a minimum from 200 to 3,000 cycles per second) freedom from singular peaks of amplification and sufficient amplification.

Of paramount importance is the actual aid that the user obtains in interpreting speech sounds and sentences correctly.

Hearing aids having a low intelligibility rating were also found to produce the greatest volume of noise by-products due to nonlinear distortion. It is concluded that these noise components are the principal cause of the poor intelligibility.

Portable hearing aids consisting of a carbon microphone and receiver only are largely ineffective because of insufficient amplification.

At present probably the best means of determining the personal usefulness of a carbon microphone hearing aid of known merit for a given person is the discrete sentence intelligibility test.

An easily performed test for eliminating the most inefficient commercial hearing aids from consideration is suggested.

Tests made with bone conduction oscillators indicated that the relative merit is not appreciably affected by the substitution of bone conduction for air conduction when carried out on persons with normal bone conduction.

TOBEY.

Intratympanic Medication of Thyroxine.

Discussion of Royal Society of Medicine, British Medical Journal, June 13, 1936.

The discussion was opened by Mr. Douglas Guthrie, who reviewed Gray's report at a previous meeting, of the treatment of fourteen cases with improvement in seven. He discussed Gray's theory that otosclerosis was the result of a defective vasomotor response in the small vessels of the ear, and suggested that local applications of thyroxine might overcome the difficulty and relieve symptoms. Mr. Guthrie believed the indiscriminate use of thyroxine, or the wrong selection of cases could only bring it into disrepute. Cases must be of recent origin and the treatment was unlikely to be successful with elderly persons. Gray had conveyed the impression that the treatment was extremely simple, but it did demand a certain degree of manipulative skill. General practitioners were now using the method and patients were coming to ask for it, therefore a warning against indiscriminate use or the notion that the procedure was quite simple should be given. Gray's former article is referred for the details of the treatment. Mr. Guthrie treated only five cases: four of them had improved in hearing, and in two the improvement was clearly demonstrated by hearing tests. None of the patients treated had complained of pain, and there was no case of hemorrhage or infection. He stated that there was a natural tendency to exaggerate the value of any treatment, especially when applied to a disease previously regarded as incurable; but he felt the injections deserved a trial in selected cases.

Results in Middlesex Hospital.—Dr. Gavin Livingstone described the results of twenty-five cases, and of these five showed an objective improvement on the audiometer. The improvement in subjective tests was frequently not confirmed by the audiometer, and the converse was occasionally true. Subjective improvement occurred in eight cases. He had injected experimentally thyroxine in the middle ear of cats. Control injection was made with flavine. The appearance of the middle ear was the same, whether flavine or thyroxine had been injected, and there was no evidence proving that thyroxine had a spe-

cific action in bringing about congestion of the mucous membrane. The speaker was doubtful whether the optimism expressed in Dr. Gray's paper would be fully justified.

Dr. Stephen Suggit gave an analysis of thirty cases treated by this method. Of these twelve showed some improvement in hearing, and out of twenty-two cases which had tinnitus six were relieved, partially or completely. The follow-up, however, had been disappointing, and no sustained improvement in hearing, subjective or objective, had been obtained. Little practical results, other than marked improvement in tinnitus in some cases, had been obtained by the injection of thyroxine. What results obtained were paralleled, if not exceeded, by a substitution of normal saline. He felt convinced that the results were due entirely to the mechanical effect of the fluid in the middle ear cavity.

Dr. T. B. Jobson described a series of fifteen cases treated. He stated that six minims appeared to be the most suitable dose. As far as hearing was concerned, there was definite improvement in one case, slight improvement in six, and no improvement in eight. Five of the seven improved cases were patients under thirty years.

Mr. R. R. Simpson gave particulars of forty cases treated by Dr. Gray's method. Recently he had sent a questionnaire to these patients. The results were: twenty-three said that the hearing had improved and thirteen that it had not; twenty-one said that their friends testified to an improvement, fourteen that they did not; twenty that the tinnitus had improved, eleven that it was the same, and three that it was worse.

A number of other men spoke on their experiences with this method of treatment with results approximately similar to those mentioned before. Mr. Douglas Guthrie, in reply, said that the one important outcome of the discussion might be that while thyroxine was not very valuable the method itself was of service, and continued experiment with other substances might lead to greater success.

GOLDSMITH-IRELAND.

Cavernous Sinus Thrombosis.

Cavenagh, J. B., British Medical Journal, June 13, 1936.

The author reports a case of cavernous sinus thrombosis with recovery. He reviews the cases from the literature that have recovered, since Eagleton's report of 1926, and discusses the various procedures used in their treatment.

A classification is made according to the type and mode of onset —for example (a) the acute fulminating, and (b) the chronic compensatory—and of the treatment according as to whether it comprises; (1) major surgery, directed at the sinus itself; (2) accessory surgical measures, dealing with the primary source of infection and draining formed abscesses; (3) serotherapy, blood transfusions, etc., in treating the general blood stream infection; or (4) spontaneous recovery without any form of treatment.

He believes the possibility of intracranial complications from Eagleton's procedure of carotid ligature have been underestimated, and this should certainly be reserved for the more desperate type of case. Blood transfusion is very much more effective if immunized donors are used and thus both the agglutinins and the bacteriolysins increased in the blood of the donor.

SUMMARY

1. A review of the cases of recovery shows no more than seven that might be described as the acute infective type.

2. Of these, three were treated by direct operation on the sinus itself and four by general blood stream therapy.

3. A combination of these two methods, with perhaps carotid ligature, is indicated if better results are to be obtained in the future.

4. Approximately thirty cases have been reported of the chronic compensatory type, and of these two have been treated by operation on the sinus itself, one by blood stream therapy, and the majority of the remainder, including the author's, by expectant treatment and by necessary surgical measures for the eradication and drainage of the primary focus, channels of invasion and formed abscesses.

5. A very few cases have recovered spontaneously.

GOLDSMITH-IRELAND.

MISCELLANEOUS

Brucellosis in Otolaryngology.

Cody, Claude C., Jr. (Houston), Arch. Otolaryng., 23:441 (April), 1936.

This disease is perhaps better known as Malta or undulent fever and may be seen in an acute fulminating form or in an ambulatory or chronic intermittent form. Stomatitis is frequently seen in the acute type, and the otolaryngologist comes in contact with the complications of brucellosis as well as with the systemic infection. Nerve deafness may occur, as with other infections. The complication of

mucosal lesions of the pharynx and larynx may resemble those of tuberculosis and syphilis. The systemic infection occasionally simulates some aspect of pyogenic infection of the ear, nose and throat. Only rarely will the need even of considering brucellosis arise except in patients with fever of uncertain origin and in atypical cases.

TOBEY.

Experimental Studies on the Action of the Saliva on Blood Coagulation in Oro-Pharyngeal Surgery (Studio sperimentale sull'azione esplicata dalla saliva sulla coagulazione del sangue in particolare riguardo alla chirurgia oro-faringea).

Battino-Viterbo, N. (Roma), Valsalva, 11:425-439 (August), 1935.

The article has an excellent review of the international literature of the subject in discussion. The experiments conducted by the author demonstrate (in vitro) that the coagulating time of the blood diminishes when the saliva is added to it, reducing it from 10 or twelve minutes to three or four minutes. This is true either for the saliva of the same or of another individual or when the saliva is mixed or filtered or centrifuged.

These experiments have also excluded the possibilities of the inorganic chemical components of the saliva, such as sulphocyanate, calcium, potassium, etc., having any influence on the coagulating time.

The saliva heated to 50 or 60 centigrade loses some of its coagulating power. For this reason it may be presumed that the saliva contains a thermostable alcohol-soluble phosphatide (cytozyene) and also a thermolabile protein or protein-like substance.

The above observation prompts the author to make the following suggestions applicable to oropharyngeal surgery:

1. Preoperative administration of atropin is contraindicated, as it stops the flow of the saliva.

2. Aspiration of the saliva during operation should be limited to the hypopharynx.

3. The gastric juice hinders the coagulating action of the saliva, therefore, sodium bicarbonate should be given if general anesthesia is administered.

4. Gargles are prohibited to prevent dilution of the saliva.

5. When the saliva is scarce, it should be stimulated by chewing hard paraffin or gum.

SCIARRETTA.

Experimental Study of the Lesions in the Mucosa of the Upper Respiratory Tract Following Partial or Total Parathyroidectomy (Contributo sperimentale allo studio delle lesioni delle mucose delle prime vie respiratorie consecutive alla paratiroidectomia totale e parziale).

Zanzucchi, G. (Parma), Arch. Ital. di Otol. Rino. and Laring., 47:313-334 (May), 1935.

The article describes histological alterations found in various tissues and organs following the disfunction of the parathyroids, mentions the possible diseases, such as otosclerosis and ozena, which may result from lack of this internal secretion, and also reviews the work of other authors on this subject.

The writer considers the hypothesis that insufficient parathyroid secretion could cause atrophic rhinitis, ozena and pharyngitis sicca, and therefore he experiments on four adult dogs and nine rabbits. Extreme care was used not to remove any tissue of the thyroid gland during the enucleation of the parathyroids. The amount of the parathyroid glands removed varied in the following order:

1. All four parathyroid glands were enucleated on two dogs and two rabbits.

2. The two external and one internal on one dogs and three rabbits.

3. The two internal from two rabbits and the two external from one dog and one rabbit.

The author concludes that the alteration found in the nasal mucosa is in direct relation with the amount of the parathyroid removed and the lapse of time transpired between the removal of the glands and the death of the animals. His histologic studies prove that:

1. In acute hypoparathyroidism there are atrophic changes, such as atrophy, disappearance of superficial epithelium, vasodilatation, vascular engorgement, cellular infiltration, etc., which increases in proportion to the duration of its existence.

2. In chronic hypoparathyroidism, in addition to the hyperemia and the cellular infiltration, there is alteration of the cylindrical epithelium and atrophy of the epithelium in the glandular tubules.

3. In the nasopharynx the epithelium becomes very thin, and in rare occasions the superficial epithelium disappears.

SCIARRETTA.